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ABSTRACT

This report contains the proceedings of the second of two hearings that explored the risks of environmental toxins to children. Testimony was heard concerning: (1) the special vulnerability of children to toxic substances; (2) exposure to lead and lead poisoning; and (3) the exposure of working children to toxins. A fact sheet gives an overview of the dangers to children of various toxins. Seven statements were delivered in person and 13 prepared statements or materials supplemental to verbal testimony were collected. Copies of various relevant documents are appended. These include: a meta-analysis of studies on low-level lead exposure and its effect on the IQ of children; a study of the prevalence and hazards of child labor; a statement on migrant farmworkers' childrens' exposure to pesticides by the Farmworker Justice Fund; a statement on environmental hazards during pregnancy by the March of Dimes; and a review of pertinent literature by the National Network To Prevent Birth Defects. (BC)

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ENVIRONMENTAL TOXINS AND CHILDREN: EXPLORING THE RISKS, PART II

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HEARING

BEFORE THE

SELECT COMMITTEE ON CHILDREN, YOUTH, AND FAMILIES HOUSE OF REPRESENTATIVES

ONE HUNDRED FIRST CONGRESS

SECOND SESSION

HEARING HELD IN WASHINGTON, DC, SEPTEMBER 13, 1990

Printed for the use of the
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ENVIRONMENTAL TOXINS AND CHILDREN: EXPLORING THE RISKS, PART II

THURSDAY, SEPTEMBER 13, 1990

House of Representatives,
Select Committee on Children, Youth, and Families,
Washington, DC.

The committee met, pursuant to notice, at 10:20 a.m., in Room 210, Cannon House Office Building, the Honorable George Miller (chairman) presiding.

Members present: Representatives Miller (chairman), Lehman, Sikorski, Durbin, Bliley, Packard, Holloway, and Walsh.

Staff present: Karabelle Pizzigati, staff director; Jill Kagan, deputy staff director; Felicia Kornbluh, research assistant; and Joan Godley, committee clerk.

Chairman **Miller**. The select committee will come to order and, again, my apologies for the time change. Between the budget summit and everything else, this place is crazy.

Let me, first of all, welcome everybody to this morning's hearing. This is the second in a series of hearings the select committee has held to take an in-depth look at environmental toxins and children.

The first hearing was held last week in Oakland, California, where we heard from parents of children, from people who work with those who have been affected by toxins, and from scientists presenting evidence to the committee about the status of children in the workplace, children in their play areas, and children where they live. We also heard about the impacts and the differential treatment that we should consider regarding children due to their exposure to toxins, to known toxins and to possible toxins, in our general environment.

This is a continuation of that investigation. As I think we are aware of, millions of children confront serious environmental risks every day. It may be in schools with asbestos-lined walls or toxic art supplies; it may be in homes tainted with carcinogenic pesticides, or painted with lead-based paint, or filled with formaldehyde.

They may be suffering secondhand effects of their parents' workplace exposure to pesticides or passive smoking, or, they themselves, as we find out with many migrant children, are directly exposed, either because they work in the fields, or because they in fact are left in the fields or brought to the fields by their parents as they seek to provide an economic livelihood for their families.

I think we have already started to see preliminary evidence that suggests we must consider children somewhat separately than we do when we look at the impact of toxins on the general population. As we have seen historically we have more or less considered the impact of toxins on adults, and not considered the impact on children.

Today, we will hear about the threat of lead and the impact of lead poisoning on young children. We are very fortunate to have Dr. Herbert Needleman who is with us this morning to discuss the latest in the long-term effects and, in some cases, irreversible effects of this very potent toxin.

As I said, this is the second in a series. We hope to be able to provide, as the select committee has been able to do on a number of different topics, without the rush of legislation or meeting legislative guidelines, to take an in-depth look at what we believe to be a serious problem confronting our nation's children.

[Opening statement of Congressman George Miller follows:]

OPENING STATEMENT OF HON. GEORGE MILLER, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF CALIFORNIA AND CHAIRMAN, SELECT COMMITTEE ON CHILDREN, YOUTH, AND FAMILIES

This morning we take a second look at the serious issues of environmental toxins and children. As we discovered last week in Oakland, California—and as we will be hearing again this morning—concern about the quality of the environment demands special consideration to the risks faced by our children.

Last week, this Committee heard tragic testimony about childhood cancer clusters in the Central Valley of California, where children of agricultural workers are exposed to large amounts of chemical pesticides.

We learned about children's exposure to pesticides from the foods they eat—including bananas, potatoes, and apples. And we learned that exposure to toxins may be more harmful to children than to their parents.

We continue that investigation today. We will take a hard look at environmental threats children face where they live, work, and learn, and evaluate their special vulnerability to these hazards.

Millions of American children confront serious environmental risks every day. They may be in schools with asbestos-lined walls or toxic art supplies. They may be in homes tainted with carcinogenic pesticides, painted with lead-based paint, and filled with formaldehyde. They may be suffering the secondhand effects of their parents' workplace exposure to lead or pesticides. Or, if they work themselves, as many migrant children do, they could be picking crops soaked with pesticides.

These children could be at risk of developmental or health problems—problems that will impair their lives, reduce their ability to learn to work productively, and cost our nation billions of dollars in health care costs.

We cannot yet claim to have all the answers. But there are some things we do know. The effects on children of exposure to *lead* are amply documented and as many as three to four million preschoolers may be lead poisoned. We are fortunate to have Dr. Herbert Needleman with us this morning to discuss his latest research on the long-term, irreversible effects of this very potent toxin.

Similarly, new research released last week charges that children exposed to environmental tobacco smoke, or "passive smoking," have a good chance of developing cancer as adults. As one witness testified in Oakland, the smoke that non-smokers, including children, take into their bodies has an even higher concentration of carcinogenic chemicals than the material inhaled by smokers directly.

On other toxins, our research base is growing. The Congressional Office of Technology Assessment recently released a report documenting the neurotoxic effects of lead and pesticides, and described the special vulnerabilities of children. Dr. Mark Schaefer will tell us about OTA's conclusions.

New research, particularly focusing on children's special risks, must be improved and expanded.

We cannot just expose problems: we must act on what we know in order to respond to the legitimate concern of parents, educators and others, who demand that children be protected from environmental dangers.

Thank you again for joining us at this important hearing.

"Environmental Toxins and Children: Exploring the Risks"

A FACT SHEET

MILLIONS OF CHILDREN VULNERABLE TO ENVIRONMENTAL TOXINS

- More than seven million of the nation's children under age 18 suffer from one or more mental disorders. Exposure to toxic substances before or after birth is one of several risk factors that appear to make certain children vulnerable to these disorders. (Office of Technology Assessment, 1990)

- The World Health Organization cites the following factors which may influence the vulnerability of children as compared with adults when exposed to chemicals: larger body surface area in relation to weight; higher metabolic rate and oxygen consumption per unit body weight; different body composition; greater energy and fluid requirements per unit body weight; special dietary needs; rapid growth during which chemicals may affect growth or become incorporated into tissues; and functionally immature organs and body systems. (World Health Organization, 1986)

MORE CHILDREN LEAD POISONED THAN PREVIOUSLY BELIEVED

- One child in six in the U.S. has dangerously elevated blood lead levels (above 10 ug/dL), including more than half of all African-American children in poverty; 400,000 newborns are delivered with toxic levels each year. (Needleman, 1990)

- Children who had elevated lead levels in their teeth at ages 6 and 7 were seven times more likely than young children with low dentin lead levels to have dropped out of school and six times more likely to have a reading disability that persisted into adolescence. (Needleman, 1990)

- Prenatal exposure to lead has been linked to delayed mental development as late as 24 months of age. At age 5, the effects of postnatal, rather than prenatal, lead exposure become pronounced. Lead exposure is associated with a range of effects from severe retardation to lower IQ, speech and language impairments, learning disabilities, and poor attention skills. (Needleman, 1990)

CHILDREN SUFFER FROM PASSIVE SMOKING

- Children of smoking parents have from 20% to 80% more respiratory problems such as wheezing, coughing, and sputum production than do children of non-smokers, as well as increased rates of chronic middle ear effusions and infections which can lead to hearing loss and consequent speech pathology. (National Academy of Sciences, 1986)

- Lung function of school-age children with smoking parents is as much as 10% lower than that of children with non-smoking parents. (Wu-Williams, 1990; Samet, 1987)

- Infants of parents who smoke have significantly more pneumonia and bronchitis than do infants of non-smokers. Studies show children of smoking parents are hospitalized for respiratory infections 20% to 70% more often than children of non-smoking parents. An estimated 8.7 to 12.4 million children are exposed to cigarette smoke in their homes. (Surgeon General, 1986; American Academy of Pediatrics, 1986)

- Studies have shown that children of smoking parents have reduced growth and development. (National Academy of Sciences, 1986)

CHILD PESTICIDE EXPOSURE MAY AFFECT LIFETIME CANCER RISK/NEUROLOGICAL DEVELOPMENT

- The average child receives four times more exposure than an adult to eight widely used cancer-causing pesticides found in food. Because of their exposure to pesticides alone, as many as 6,200 children may develop cancer sometime in their lives. More than 50% of the lifetime cancer risk from carcinogenic pesticides used on fruit is estimated to occur during a child's preschool years. (Natural Resources Defense Council, 1989)

- From 17% to 58% of the country's 18 million children ages 1 to 5 are being exposed to neurotoxic organophosphate pesticides at levels above what the federal government considers safe. (Natural Resources Defense Council, 1989)

- Toxic substances, such as lead and organochlorine pesticides like DDT, are known to be present in breast milk and are transferred to the nursing child. The amount of toxic substances in a breastfeeding child can surpass levels in the mother's body. (Wolff, 1990)

HOME PESTICIDE USE PLACES CHILDREN AT RISK OF ILLNESS

- In Dallas, Texas, a review of 37 hospitalized pesticide poisonings among infants and children at the Children's Medical Center revealed five cases were due to pesticide exposure from playing on carpets and floors of homes following spraying or fogging inside residences. (Zwiener, 1988)

- Six of 21 children admitted to Arkansas Children's Hospital for organophosphate poisoning were judged to have been exposed following insecticide spraying inside the home. (Fenske, 1990)

- Parental use of pesticides both in the home and in the garden may increase the risk of childhood leukemia as much as seven-fold. (Lowengart, 1987)

CALIFORNIA CHILD CANCER CLUSTERS/BIRTH DEFECTS RAISE CONCERN

- In the agricultural community of McFarland, California (population 6,400), ten cases of cancer in children under 20 were observed from 1975 to 1985 when three cases would have been expected. From 1982 to 1985, when one case would have been expected, eight were observed. (Kern County Health Department, 1986)

- In Earlimart, California (population 4,414), five cases of childhood cancer were observed from 1986 to 1989 when only 0.4 cases would have been expected based on the National Cancer Institute SEER (Surveillance Epidemiology and End Results) data for Hispanics. All of the parents of these children are farmworkers and the mothers of four of the children worked in the grape vineyards during their pregnancy. (Moses, 1989)

- Children born in areas with high pesticide use are twice as likely to be born with limb reduction defects than children born in areas of minimal pesticide use. (Schwartz, 1988)

At this time, I would like to recognize the ranking minority member, Mr. Bliley of Virginia.

Mr. BLILEY. Thank you, Mr. Chairman.

Today we confront the fearful symmetry of science. While the modern world beckons science to eradicate disease, feed five billion people, and build the infrastructures necessary to sustain a high quality of living, scientific advances have also brought new threats to our health and safety.

There are many examples of our bittersweet relationship with science. Powerful drugs, which should be used to heal, are abused for self-gratification and cause further damage. Energy sources which free us to engage in fruitful commerce also trap us into dangerous dependency. Developing nations which need to industrialize to lift their people out of poverty may not discover until it is too late that they may be mortgaging their future by failing to protect the environment.

Science nearly always has both immediate and long-term effects. It is more than obvious that there are both benefits and risks in the application of science. It would be absurd to hold a referendum to ban any substance that is a toxin in a zeal to protect children. Such an extreme would eliminate all modern medicines and close every hospital, as well as make food so expensive that only the most well-to-do could afford the most nutritious fresh commodities.

Reformers should not fail to recognize that children from low-income families would suffer disproportionately from the effects of banning the use of synthetics for crop production. It is difficult not to overreact when it comes to the protection of our children, but, as policymakers, we must avoid perverse and unintended outcomes. Science has provided government with powerful tools such as risk assessment, and we should use this information to improve public confidence in decision-making.

We must not undermine public confidence by pretending that government regulation is nonexistent. Federal regulations impose direct costs on the economy of roughly $175 billion per year or more than $1,700 for every taxpayer in the United States. It is estimated that $100 billion of this amount is due to regulations on environmental hazards. Congress must assure that the executive branch has the tools it needs to set appropriate standards.

While all of us in Congress take the bureaucracy to task at one time or another for lax enforcement, we must also acknowledge that the overall level of enforcement activities is substantial. EPA administrative actions under the Toxic Substance Control Act, for example, have increased from less than 100 in fiscal year 1980 to more than 500 in fiscal year 1989. Over $28 million has been assessed in administrative penalties under this act alone.

The states share responsibility for environmental enforcement and issued more than 12,000 administrative actions to violators in 1989 and referred over 700 civil cases to states attorneys general.

There is a tendency to conjure up mental images which make the present time appear in a worse light. We should be careful, however, not to fall into this public relations trap. Historical data shows that lead emissions have been reduced from nearly 204,000 tons per year in 1976 to 8,000 tons per year in 1987. Carbon monoxide emissions have been reduced by nearly 40 percent in the past 20 years.

In terms of public health, we find that the death rate for malignant tumors for children ages 1 to 4 has declined from 11.7 per 100,000 in 1950, to 4.5 per 100,000 in 1980, to 3.8 per 100,000 in 1987. For children between the ages of 5 and 14, the death rates have been cut in half since 1950, including a 23 percent reduction since 1980.

The cases of occupation-related skin disease or disorders has declined from 65,900 in 1978 to 54,200 in 1987. Although the cancer incidence rates continue to rise, there are some hopeful trends. The rate of increase has been slowed for white males and black females, and the cancer rate for black males has actually declined since 1983.

None of this is to say that we cannot make further improvements. However, changes in public policy require solid evidence that there is a clear risk which can be reduced in proportion to the cost of further regulation. Does such evidence exist? Does the risk require additional federal regulation and oversight, or are there less costly alternatives which will remedy the problem? Does the proposed remedy actually increase other health risks? These are some of the questions which should be fully explored.

Science should not be exploited for political gain by dividing people into an "us versus them" issue. Assuring the safety of our food, workplace, and schools demands cooperation, not a needless sense of hopelessness. Everyone concerned, employer, worker, manufacturer, government, and consumer, has a vital stake in properly identifying a problem and working together to find the solution.

The employer has a responsibility to ensure that the worker is appropriately trained to handle chemicals safely. The worker is responsible for adhering to safety procedures. The manufacturer is obligated to meet government regulations strictly. The consumer must become educated about those simple, daily tasks which also reduce risks. All of these members are parts of the same body. To purposely create fear and panic will ultimately prove to cause more damage than good.

I thank you, Mr. Chairman, for your indulgence. I apologize for the length of the statement.

[Opening statement of Congressman Thomas J. Bliley, Jr. follows:]

Opening Statement of Hon. Thomas J. Bliley, Jr., a Representative in Congress From the State of Virginia and Ranking Republican Member

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ENVIRONMENTAL TOXINS AND CHILDREN:
EXPLORING THE RISKS

Republican Fact Sheet

September 13, 1990

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PESTICIDES

Little is known about the extent or magnitude of chronic health problems related to occupational exposure to pesticides because appropriate studies have not been done. ["Pesticide-Related Health Problems and Farmworkers," Marion Moses, AAOHN Journal, March 1989, p. 119.]

Results observed in treated and untreated plots...suggest that, without insecticide treatment, insect losses alone would average about 45 percent.... ["Pesticides: Assessing the Risks and Benefits," Chris F. Wilkinson, May 1990, p. 5.]

...no more than 30-40 pesticide-related deaths occur annually in the U.S. and the majority of these involve suicide and accidents associated with incompetence or gross safety violations. Unfortunately, few if any, epidemiological or other data exist to support any relationship between occupational exposure and adverse chronic effects on human health. ["Pesticides: Assessing the Risks and Benefits," Chris F. Wilkinson, May 1990, p. 9.]

Only the state of California requires mandatory reporting of pesticide-related illness, with 1,211 cases in 1986. (CA Dept. Ag., 1987.) However, the California system is based on doctor reporting through the workers' compensation system. Many affected workers never see a doctor, are not properly diagnosed or are unaware of their rights under the law. The most frequently mistaken diagnoses in workers with pesticide poisoning are flu and gastroenteritis. ["Pesticide-Related Health Problems and Farmworkers," Marion Moses, AAOHN Journal, March 1989, p. 117.]

PESTICIDE RESIDUES IN FOOD

The American Academy of Pediatrics recognizes that the risks for pesticides in the diet are remote, long-term, and theoretical and there is no cause for immediate concern by parents. ["Pesticide Residue in the Diet of Children," AAP News, April 1989, p. 10.]

One major group of natural chemicals in the human diet are the chemicals that plants produce to defend themselves, the natural pesticides. We calculate that 99.99% (by weight) of the pesticides in our diet are natural. ["Too Many Rodent Carcinogens," Bruce N. Ames and Lois Swirsky Gold, Science, Aug. 31, 1990.]

My own estimate for the number of cases of cancer or birth defects caused by man-made pesticide residues in food or water pollution-usually at levels of thousands or millions of times below that given to rats or mice- is close to zero. ["Be Wary of Nature's Own Pesticides," Bruce Ames, L.A. Times, Feb. 27, 1989, p. 5.]

In order to minimize cancer & the other degenerative diseases of aging, we need the knowledge that will come from further basic scientific research. Yet we are spending $70 billion per year on pollution because of widely exaggerated fears & only $9 billion per year on all of our basic scientific research. ["Be Wary of Nature's Own Pesticides," Bruce Ames, L.A. Times, Feb. 27, 1989, p. 5.]

LEAD

Lead is a toxin that affects every system in the body. It is particularly harmful to the developing brain and nervous system, so that lead exposure is especially devastating to the fetus and young children. Very severe lead exposure can cause coma, convulsions, and even death. Lower levels of lead, which usually do not cause symptoms, result in decreased intelligence, decreased ability to learn, developmental disabilities, behavioral disturbances, and disorders of blood production. [Testimony of Vernon N. Houk, M.D., Assistant Surgeon General, Centers for Disease Control, before the Subcommittee on Toxic Substances, Environmental Oversight, Research and Development, March 8, 1990, p. 2.]

In the past two decades, knowledge of the effects of lead poisoning has changed substantially. When national childhood lead poisoning prevention programs were instituted in the early 1970s, lead encephalopathy and other manifestations of severe overt lead poisoning were common. Today these outcomes are rare--to a great extent because of childhood lead-screening programs in high-risk areas and reduction of lead in the environment (particularly for gasoline, air, and food). ["Childhood Lead Poisoning--US: Report to the Congress by the Agency for Toxic Substances and Disease Registry," in *JAMA*, September 16, 1988, p. 1533.]

In FY 1981, the last year of CDC management, 535,730 children were screened, with a positive toxicity rate of 4.1%, or 21,897 children. In FY 1983, reports from the state agencies indicated that 676,571 children were screened, and 9,317, or 1.6%, had elevated lead exposure of less than 30 mg/dL....in December 1986, 785,285 children were screened in about 40 programs. Of these, 11,739 children, or 1.5%, had elevated Pb-B levels that met CDC's toxicity classification. [*The Nature and Extent of Lead Poisoning in Children in the US: A Report to Congress*, Agency for Toxic Substances and Disease Registry, PHS, 1988, p. I-13.]

Valid estimates of the total number of lead-exposed children according to standard metropolitan statistical areas (SMSAs) or other appropriate geographic units smaller than the nation as a whole are not possible. ["Childhood Lead Poisoning--US: Report to the Congress by the Agency for Toxic Substances and Disease Registry," in *JAMA*, September 16, 1988, p. 1523.]

Since the estimated numbers of children for each source and category are not comparable, they cannot be used to rank the severity of the lead problem by source of exposure. ["Childhood Lead Poisoning--US: Report to the Congress by the Agency for Toxic Substances and Disease Registry," in *JAMA*, September 16, 1988, p. 1529.]

Although data are very limited, an estimated 233,000 children are exposed to lead from stationary sources of all types. ["Childhood Lead Poisoning--US: Report to the Congress by the Agency for Toxic Substances and Disease Registry," in *JAMA*, September 16, 1988, p. 1529.]

Lead has no biologic value. Thus, the ideal whole blood lead level is 0 mg/dL. According to the NHANES II, conducted from 1976 to 1980, the mean blood lead level in American preschool children was approximately 16 mg/dL. ["Statement on Childhood Lead Poisoning," Committee on Environmental Hazards and Committee on Accident and Poison Prevention, in Pediatrics, March 1987, p. 457.]

Between 1976 and 1980, the average blood lead level in Americans of all ages decreased from 15.8 to 10.0 mg/dL according to the NHANES II. This decrease coincided with a reduction in the use of lead additives in gasoline. ["Statement on Childhood Lead Poisoning," Committee on Environmental Hazards and Committee on Accident and Poison Prevention, in Pediatrics, March 1987, p. 458.]

HOUSEHOLD POLLUTION

Cooking produces about 2000 mg per person per day of mostly untested burnt material that contains many rodent carcinogens....The total amount of browned and burnt material consumed per person in a typical day is at least several hundred times more than that inhaled in a day from severe outdoor air pollution....the intake of these carcinogenic nitropyrenes has been estimated to be much higher from grilled chicken than from air pollution. ["Environmental Pollution and Cancer: Some Misconceptions," Bruce N. Ames and Lois Swirsky Gold, in Science and the Law, Peter Huber, Ed., p. 8.]

It is important to note that in no studies has a child's risk of lung cancer developing in adulthood after exposure to radon in the home environment been examined. It has yet to be determined whether the risks of lung cancer derived from studies of men who were occupationally exposed to radon in the underground mines apply to children. ["Radon Exposure: A Hazard to Children," Committee on Environmental Hazards, American Academy of Pediatrics, Pediatrics, May 1989, p. 800.]

Most of our knowledge about the health hazards of radon exposure comes from studies of heavily exposed uranium miners....The health effects of low levels of radon like those found in homes have not been studied. We don't know whether health risks for women or children could be the same as those for miners, who are generally healthy males.... ["Radon and Human Health: Research Supported by the National Institute of Environmental Health Sciences, NIH, October 1988, p. 2.]

ASBESTOS

Asbestos-induced cancer and asbestosis are diseases that are almost never seen in children. Practicing pediatricians should not expect to see any asbestos-related disease in their patients. ["Asbestos Exposure in Schools," Committee on Environmental Hazards, Pediatrics, February 1987, p. 301.]

There are no available quantitative data on risk at the levels of airborne asbestos found in schools....Asbestosis is unlikely to occur after exposure such as in schools. ["Asbestos Exposure in Schools," Committee on Environmental Hazards, Pediatrics, February 1987, p. 301 and 302.]

The extent of danger cannot easily be quantified because, while

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EPA is of the opinion that any level of exposure to asbestos involves some health risk, the exact degree of risk cannot be reliably estimated. ["Asbestos in Schools: Low Marks for Government Action," Robert D. Lang, Environment, November 1984, p.17.]

MULTIPLE CHEMICAL SENSITIVITIES

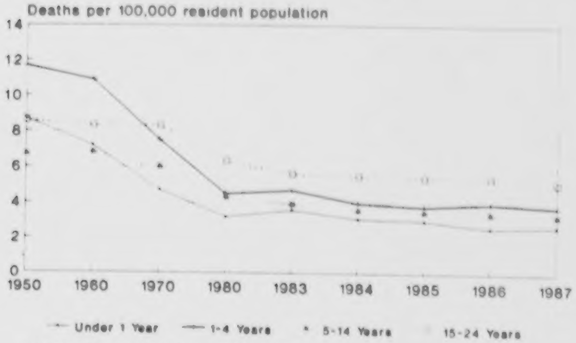
The Board on Environmental Studies and Toxicology of the National Research Council, the research branch of the National Academy of Sciences, estimates that 15% of the population experiences hypersensitivity to chemicals found in common household products. ["Multiple Chemical Sensitivities (MCS)," Linda Lee Davidoff, The Amicus Journal, Winter 1989, p. 15.]

The basic mechanism of hypersensitivity reactions are not yet understood. ["Multiple Chemical Sensitivities (MCS)", Linda Lee Davidoff, The Amicus Journal, Winter 1989, p. 16.]

ENVIRONMENT AND CANCER

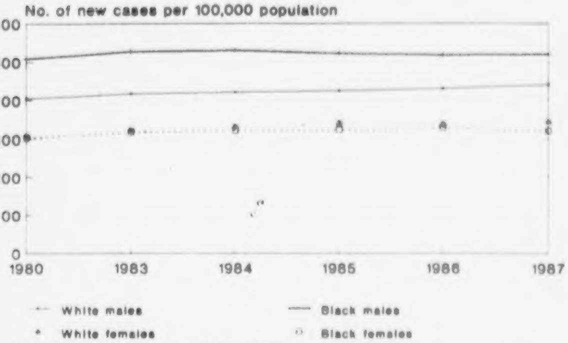
There is no persuasive evidence that life in the modern industrial world has in general contributed to cancer deaths....Although the statistics are less adequate on birth defects, there is no evidence that they are increasing. Conclusion: Americans are healthier now than they have been in their history. ["Environmental Pollution and Cancer: Some Misconceptions," Bruce N. Ames and Lois Swirsky Gold, in Science and the Law, Peter Huber, Ed., p. 2.]

Death Rates for Malignant Tumors According to Age



Health United States 1989, HHS

The graph above shows that the death rate for malignant tumors has dropped considerably over the past four decades for children and young adults. The overall decline in the death rate due to tumors for this group has continued throughout the 1980s.



Cancer Incidence Rates According to Sex and Race

Health United States, 1989, HHS.

The rates represented in this graph show that the incidence of cancer among white/black males and females of all age groups has stayed relatively constant since 1980. The last decade has seen a stable cancer rate with no significant increases.

FEDERAL REGULATORY FRAMEWORK REGARDING TOXINS

According to a 1990 report by the Office of Technology Assessment (OTA), it is the responsibility of regulatory agencies to limit public exposure to toxic chemicals through programs mandated by law. Because of the great diversity of toxic substances, many statutes exist to control their use.

The federal regulatory structure has been established by four major acts:

1. Toxic Substances Control Act (TSCA)
2. Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)
3. the Federal Food, Drug and Cosmetic Act
4. Occupational Safety and Health Act

The following federal agencies have jurisdiction over laws (cited below) setting federal guidelines and/or research grants dealing with environmental toxins and other hazards.

ENVIRONMENTAL PROTECTION AGENCY

Responsible for implementing two of the major acts, TSCA and FIFRA. EPA has a large intramural research program devoted to environmental neurotoxicology.

FOOD AND DRUG ADMINISTRATION

The Federal Food, Drug and Cosmetic Act covers a wide range of substances. It authorizes FDA to require submission of specific toxicity test data before permitting food additives, drugs and other substances to be marketed.

Research programs within FDA are conducted at the National Center for Toxicological Research (NCTR) in Jefferson, Arkansas, and at the Center for Food Safety and Applied Nutrition in Washington, D.C.

CONSUMER PRODUCT SAFETY COMMISSION

CPSC is an independent regulatory commission charged with protecting the public from "unreasonable risks" of injury associated with consumer products." Risk of injury is defined as "risk of death, personal injury, or serious or frequent illness."

DEPARTMENT OF HOUSING AND URBAN DEVELOPMENT

The Lead-Based Paint poisoning Prevention Act of 1971 required that the HUD eliminate as far as practicable the hazards of lead paint in existing houses, and mandated that the Department promulgate necessary regulations.

NATIONAL INSTITUTES OF HEALTH

NIH supported more than 200 neurotoxicology-related research projects in fiscal year 1988. Most of the projects were extramural competitive grants to investigators in public and private institutions.

ALCOHOL DRUG ABUSE AND MENTAL HEALTH ADMINISTRATION

ADAMHA funds extensive neurotoxicity research at all three of its Institutes. The National Institute for Drug Abuse (NIDA) and the National Institute for Mental Health (NIMH), for example, both funded a large number of extramural research grants.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

NIOSH, located within Centers for Disease Control (CDC), has identified neurotoxic disorders as one of the Nation's top 10 leading causes of work-related disease and injury.

[Neurotoxicity: Identifying and Controlling Poisons of the Nervous System (Summary), Congress of the United States, Office of Technology Assessment, April 1990.]

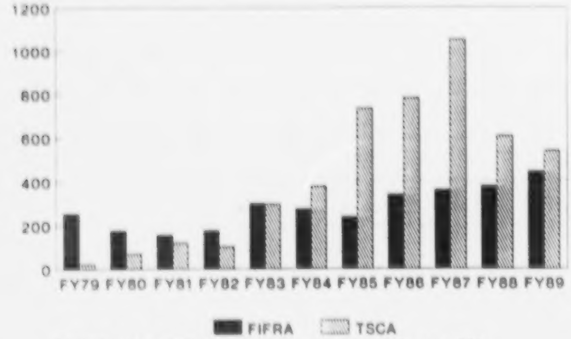
ENFORCEMENT OF FEDERAL LAWS AND PENALTIES ASSESSED

Since the beginning of the Environmental Protection Agency in 1970, the EPA has imposed a total of $185.9 million in civil penalties ($128.8 million with civil judicial actions and $57.1 million with administrative actions). "In FY 1989, $34.9 million in civil penalties were assessed, $21.3 million in civil judicial penalties (the second highest total in the Agency's history) and $13.6 million in administrative penalties (an all-time record)."

EPA has imposed over $28.5 million in Toxic Substances Control Act civil administrative penalties. The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and Safe Drinking Water Act programs are primarily enforced by the States; however, EPA has levied $2.4 million and $1.5 million under these statutes, respectively.

[EPA Enforcement Accomplishments Report: FY 1989, p. 18.]

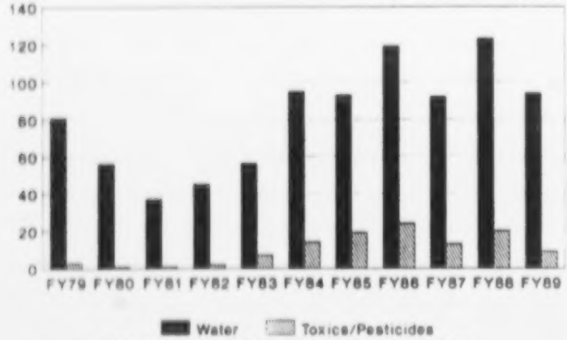
EPA Administrative Actions Initiated
1979-1989



Source: 1989 Enforcement Accomplishments Report, EPA.

The EPA set a record high number for administrative enforcement in FY 1989 with just over 4,000 actions taken. The chart above shows the administrative actions undertaken by just two of six major EPA acts since 1979.

The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), is the major federal law dealing with pesticides. The Toxic Substances Control Act (TSCA), is concerned with the use of hazardous chemicals.



EPA Civil Referrals to the Department of Justice

Source: 1989 Enforcement Accomplishments Report, EPA.

The EPA referrals to the Department of Justice were at an all-time high in 1988 with 372 cases. The cases regarding water and toxic/pesticides violations referred to DOJ between FY 1979 to FY 1989 are shown on the chart.

FEDERAL LAWS RELATED TO EXPOSURE TO TOXIC SUBSTANCES

There are more than 20 major pieces of federal legislation related to exposure to toxic substances which fall under many of the agencies listed above. This list does not include state laws or initiatives being proposed.

1. Food, Drug and Cosmetics Act (1906, 1938, amended 1958, 1960, 1962, 1968, 1976)
   Agency: Food and Drug Administration
   ** Food, drugs, cosmetics, food additives, color additives, new drugs, animal and feed additives, and medical devices.

2. Federal Insecticide, Fungicide and Rodenticide Act (The Pesticide Act) (1948, amended 1972, 1975, 1978, 1988)
   Agency: Environmental Protection Agency (EPA)
   ** Pesticides

3. Dangerous Cargo Act (1952)
Agency: Department of Transportation, United States Coast Guard
** Water shipment of toxic materials

4. Atomic Energy Act (1954)
Agency: Nuclear Regulatory Commission (NRC)
** Radioactive substances

5. Federal Hazardous Substances Labeling Act (1960, amended 1981, 1988)
Agency: Consumer Product Safety Commission (CPSC)
** Labeling of toxic household products and art materials

6. Federal Meat Inspection Act (1967) Poultry Products Inspection Act (1968)
Agency: United States Department of Agriculture (USDA)
** Food, feed, color additives and pesticide residues.

7. Egg Products Inspection Act (1970)

8. Occupational Safety and Health Act (1970)
Agency: Occupational Safety and Health Administration (OSHA) and National Institute for Occupational Safety and Health (NIOSH)
** Workplace toxic chemicals

9. Poison Prevention Packaging Act (1970, amended 1981)
Agency: CPSC
** Packaging of hazardous household products and child resistant closures on toxic hazardous materials (16 categories)

10. Clean Air Act (1970, amended 1974, 1977, 1987, in conference 1990)
Agency: EPA
** Air pollutants

11. Hazardous Materials Transportation Act (1972)
Agency: DOT
** Transport of hazardous materials

12. Clean Water Act (formerly Federal Water Control Act) (1972, amended 1977, 1978, 1987)
Agency: EPA
** Water pollutants

13. Marine Protection, Research and Sanctuaries Act (1972, 1988)
Agency: EPA
** Ocean dumping

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14. Consumer Product Safety Act (1972, amended 1981)
Agency: CPSC
** Hazardous consumer products

15. Lead-based Paint Poison Prevention Act (1973, amended 1976)
Agency: CPSC, Department of Health and Human Services, Housing and Urban Development (HUD)
** Use of lead paint in federally assisted housing.
CPSC allows no more than .06 percent of lead in paint, excluding marine paints (for ships), street paints and some other types of industrial paints.

16. Safe Drinking Water Act (1974, amended 1977, 1986, 1988)
Agency: EPA
** Drinking water contaminants. In 1988 amended by Lead Contamination and Control Act.

17. Lead Contamination Control Act (1988 amendment to Safe Drinking Water Act)
Agency: EPA
** Issued a ban on the manufacture and sale of water coolers and fountains with lead lined tanks.

18. Resource Conservation and Recovery Act (1976, amended 1980, 1984, 1988)
Agency: EPA
** Solid waste, including hazardous waste. Amended in 1988 by Medical Waste Tracking Act.

19. Toxic Substances Control Act (1976, 1986, 1988)
Agency: EPA
** Hazardous chemicals not covered by other laws, includes pre-market review.

20. Asbestos Hazard Emergency Response Act (1986)(amendment to Toxic Substances Control Act)
Agency: EPA
**Requires that all primary and secondary public and private schools be inspected for asbestos and manage asbestos.

21. Federal Mine Safety and Health Act (1977)
Agency: Department of Labor, NIOSH
** Toxic substances in coal and other mines

22. Comprehensive Environmental Response, Compensation, and Liability Act (i.e., Superfund) (1981, amended 1986 by the Superfund Amendments and Reauthorization Act)
Agency: EPA
** Hazardous substances, pollutants and contaminants.

23. Asbestos School Hazard and Abatement Act (1984)
Agency: EPA
** Provides federal grants and loans to public and private schools for asbestos abatement.

24. Indoor Radon Abatement Act (1988)
Agency: EPA
** Provides technical assistance and grant assistance to states for radon programs and the study of radon in schools.

["Chemical Carcinogens: A Review of the Science and Its Associated Principles," Environmental Health Perspectives. U.S. Interagency Staff Group on Carcinogens. Ronald W. Hart, Chairman, National Center for Toxicological Research, Food and Drug Administration. Vol. 67, pp. 201-202, 1986; Selected Environmental Law Statutes, West Publishing, 1989-90; CRS Report for Congress, "Summaries of Environmental Laws Administered by the Environmental Protection Agency," March 27, 1989, pp. 1-52.]

Chairman **Miller**. Mr. Sikorski.

Mr. **Sikorski**. Thank you, Mr. Chairman.

I am not a scientist or researcher, an oncologist or biologist or horticulturalist; I am a father, a concerned and a little scared father because of the environmental dangers my child must face every day at home, at school, in between. Dwight Eisenhower said something that could be considered profound. He said, "Things were different before they changed."

Like most of my generation, I was raised with certain embroidery-like, clear, clean, life-living principles, and for me they were drawn, I suppose, from Dr. Spock, and Yul Gibbons, and "I Love Lucy," and "Captain Kangaroo"; such as, an apple a day keeps the doctor away; government is truly looking out for Americans, especially America's children, and protecting our food supply; that the Environmental Protection Agency is just that; that the Food and Drug Administration is testing food and drugs and guarding our food supply; that chemical companies wouldn't risk or wouldn't even want to market something that might be harmful or dangerous to people; and that kids should drink that refreshing, cool, clear water coming out of their drinking fountains at school—it's a lot better than the Kool-Aid or the pop drinks.

Well, we know none of these are absolute facts. It is not easy raising a kid in today's society. There is ALAR in their apples, and radon in the basement, and lead in their drinking water, and asbestos in their schools. I want to look at a couple of these issues.

We are going to hear from some of the witnesses a little rehash of the old ALAR business. Most of us know that ALAR, daminozide, breaks down as UDMH, and, prior to the highly-publicized debate, almost no one knew this. Certainly, no one told me that a chemical compound used also as a rocket fuel additive was being sprayed on trees that fed my daughter and also was found to cause tumors in laboratory animals in four independent studies.

I am a little angry because all the time I was feeding my daughter food that I thought was good for her. It was her first solid food. It was applesauce. And we poured more apple juice in her than gas in a race car. And I know I am not alone; most of the parents in my generation did the same thing. I know ALAR is not the only offender; in fact, it is not the big offender.

There are hundreds of pesticides on the market today that leave residue on our foods and that we are unsure whether they are safe or what their tolerance levels truly are. We don't know what they do to children, and we don't know what they do together as they accumulate on food and are fed to our children.

What we are sure of is that our children are much more susceptible to toxins than we are; that our children don't have the same developed systems to move these toxins out of their bodies; and, when they are left in their bodies, children have undeveloped immunological systems that perhaps cannot fight them off; that our children have different growth patterns, different eating patterns than we do.

EPA has ignored these differences. In the past 35 years, tolerance levels for pesticides on food that goes to our kids have been set not for the most vulnerable segments of our population, including our children, but for the average diet and tolerance of an 18-

year-old white male. The tolerance levels have been set to protect economic interests above public health interests.

In fact, most of the toxins used on food and most of our fresh food has several toxins, 10 or 20 approved for each one of them, each of them have not been reviewed or had modern tolerance levels set. Most were grandfathered in in 1972, and we're living in a "don't worry, be happy age." When you go to a grocery store or hear that something meets the requirements set by the EPA, there may be no requirements at all, and, likely, they were set in 1972 by a political decision to grandfather these chemicals in.

Likewise, less than 1 percent of the food comes to us as tested by the Food and Drug Administration, and less than 50 percent of the high-risk pesticides are even tested for.

We are going to hear about lead. In the past couple decades, we have acted to control the environmental threat of lead to our children. Lead in gasoline is being phased out. Lead in paint and baby food tins has been banned altogether. Lead in the air has been reduced by 80 percent, and the average level in human blood fell 37 percent between 1976 and 1980.

Nevertheless, until recently, we missed a major source of lead poisoning, the water we drink. As a result, millions of Americans, particularly kids, again, remain at risk. Every year a quarter of a million American kids suffer an IQ loss due to lead in their drinking water.

Seventeen percent of all America's kids, more than three million under the age of seven, have levels of lead that are neurotoxic. One in 11 of America's children under age 6, 1.5 million kids, have blood levels that meet the U.S. Centers for Disease Control's definition of acute lead poisoning. The American Academy of Pediatrics has identified lead as the most serious toxicological threat to America's children.

In the 100th Congress I authored legislation that President Reagan signed to reach school children by testing kids for elevated blood levels and removing sources of lead in school drinking water systems and going after lead in the drinking fountains. We have not funded that fully. I recently introduced the Lead Pollution Control Act, which will be the subject of a congressional hearing next week.

I have more, but I urge parents to keep informed, keep their children healthy, and, above all, keep the pressure on all of us, the scientists, the public policymakers, the managers, and the politicians. Because if we cannot direct and fund the agencies to do serious monitoring, real testing to prevent the pesticide and lead exposure of our kids, we don't belong in business as the government of the United States of America.

Thank you, Mr. Chairman.

Chairman **MILLER**. Congressman Packard.

Mr. **PACKARD**. Thank you, Mr. Chairman.

I will be short. I want to thank you for holding these hearings. We have all become more aware of the state of our environment and the cleanliness of our air. The issue of toxins is one that needs to be addressed. There are many agencies which we already have that have responsibility to regulate and to study exposure levels of toxins. We already have 20 laws regulating toxins. In addition, many states, such as my own in California, have also enacted tough, restrictive legislation of these toxic substances.

However, I am concerned that we have not completely studied the issue. We know there is a correlation between toxins and the air quality. We have not determined the levels which are dangerous to the extent that we should and particularly as it relates to children. I believe that the effect that toxins have on children is worthy of our further attention. I do not believe, however, that we should rush to judgment without hard data and back-up information.

It may be a coincidence, but here in front of me is a roach-motel, and it says on the back, "Keep out of reach of children."

Chairman **Miller**. It wasn't meant for members of the committee.

Mr. **Packard**. That may be—well, I'll rest my case.

Chairman **Miller**. Mr. Walsh.

Mr. **Walsh**. Thank you, Mr. Chairman. I thank you for holding this hearing.

I think it is very, very important that we deal with the facts instead of conjecture and nonscientific debate. I am a member also of the Agriculture Committee, and we have spent a lot of time talking about things like pesticides and chemicals used in agriculture. I would caution everyone that no one, to our knowledge, has ever died of pesticides unless they took them straight. There have been incidents, apparently, of individuals taking pesticides, ingesting them, and dying from them. Certainly, they are deadly poisonous when you do that.

ALAR, the chemical ALAR, there is no question that it is carcinogenic. I worked on an apple orchard when I was a young man and did not realize it at the time, but we were using it. It was called "sticker" in those days. What it does is, it makes the apples hang on the tree a little bit longer and ripen and not fall off. And it is used only in northern climes.

Most apple orchards today do not use it. Today none use it. Last year some used it in the farther northern reaches of New York State where you have a shorter growing season. And it is used only on McIntosh apples, and McIntosh apples make up less than 10 percent of the total U.S. apple crop.

So there were a lot of people very scared about ALAR. In fact, there was very, very little risk that anyone would have eaten an apple with ALAR in it or on it and even less risk that that chemical would have affected them in any way. However, I am just as concerned as everyone else that we make sure that our systems, governmental systems, seek those chemicals out, and if the EPA has evidence that they are carcinogenic, they should be removed immediately. I have supported legislation to do that.

I am reminded of an argument that was posed when my dad was involved in government up home in Central New York. They were going to put fluoride in the water. Fluoride would prevent tooth decay and keep your teeth stronger, healthier, happier, and so on.

An individual showed up at a hearing with a jar of fluoride, and he held it up, and he said, "Look at this." It was not a jar of fluoride; it was rat poison. He held up this rat poison, and it said right on it, it says right here, "Fluoride. Kills rats." Well, I suppose if you took the rat poison, the fluoride would probably get you, but if you put it into water in very, very tiny little doses, it has a beneficial effect.

What we have to make sure of—and I guess I am carrying this point maybe too far—but let's deal with the facts. Let's not scare people. There were people, after the "60 Minutes" story, who were calling up the state troopers to stop the school bus because their child had an apple in their lunch bag.

We have to be very, very careful. We do have the purest, best, safest, and cheapest food supply in the world. Chemicals, believe it or not, have something to do with that. Let's just make sure that we are very, very clear that we are dealing with facts and not drum up any more hysteria to scare people.

I do believe that there are certain things that we should be very, very concerned about, lead poisoning, and so on, and I am very interested in hearing what the expert witnesses have to say.

Thank you, Mr. Chairman.

Chairman **MILLER**. Congressman Lehman.

Mr. **LEHMAN**. I am just here to listen, Mr. Chairman.

Chairman **MILLER**. With that, we will begin with the witnesses. The first panel we will hear from is made up of Nancy Greenspan, who is a parent from Bethesda, Maryland; Dr. Mark Schaefer, who is a project director from the Office of Technology Assessment, who will be accompanied by Dr. Roger Herdman, assistant director, Office of Technology Assessment; Dr. Herbert Needleman, who is a professor of psychiatry and pediatrics, University of Pittsburgh, School of Medicine; and Richard Wilson, who is a professor of physics from Harvard University.

If you would, please come forward. Your written statements and whatever supporting documents you want to provide for the committee will be made part of the record in their entirety. The extent to which you can, you may summarize, so it will allow time for questions.

We will begin, Mrs. Greenspan, with you. Thank you very much for joining the committee this morning, and we appreciate all of the help that you all have provided.

STATEMENT OF NANCY GREENSPAN, PARENT, BETHESDA, MARYLAND

Mrs. **GREENSPAN**. Thank you, Mr. Chairman.

Committee members, ladies and gentlemen, my name is Nancy Greenspan. I am here as the mother of three children, one of whom has a chronic illness, and also as the co-founder and director of a local environmental group.

Last year, in recognition of research on the growing link between environmental toxins and chronic illness, the National Academy of Sciences organized a panel to review this subject. It is one that I have been dealing with since my daughter Sarah was diagnosed with juvenile diabetes at the age of two.

For the last six years, my husband and I have been carefully monitoring Sarah's glucose levels, fluid intake, and general environment. Using this information, we have learned that her reaction to chemicals has a dramatic impact on the control of her diabetes. We discovered that petrochemicals, solvents, commercially-produced meats (those containing large amounts of growth hormones and antibiotics) profoundly influence her glucose levels and her physical comfort. An aid in separating out these effects was Sarah's lack of need of insulin in her first year after diagnosis. Three short examples illustrate the effects of chemicals on Sarah.

Our first experience with chemical reactions occurred on three consecutive Tuesday evenings when Sarah suddenly had high glucose levels after dinner—an average of 175 rather than the usual 85 to 100. During each of these weeks, these high levels gradually subsided so that by Sunday she was again in the normal range.

On the Monday morning of the fourth week, I came home to find the cleaning lady mopping the kitchen floor with water and ammonia, a product we had removed from the house because of general concern with household chemicals. With Sarah starting nursery school, the cleaning lady had thought it was safe to use ammonia again. Eliminating the ammonia, and no other change, broke the pattern, and Sarah's glucose levels returned to normal.

Over time, as other sudden and mysterious reactions developed, chemicals always seemed to be a factor. We were able to resolve one episode of fluctuating glucose levels by removing natural gas from our house. Whenever Sarah spent any time in one particular room, her levels were always higher.

On a hunch, we had the gas company come and check the pipes. In the part of the basement just below this room, the repairman found a leak. While removing the gas lines from the house, Sarah and I moved to an understanding neighbor's house which did not contain any gas pipes, and Sarah was fine.

Although Sarah's serious reactions are mostly confined to inhalants—with strong reactions to substances such as pesticides and industrial cleaners—she can have similar reactions to foods containing chemical additives.

About six months after we finally started Sarah on insulin—she was three and a half at this time—she fell into a pattern of sleep problems, lethargy, and chronic hunger. Equally troubling, her glucose fasting level doubled. Poring over our records, we noted that her fasting time levels were higher on those mornings following nighttime snacks of meat protein.

Months before, we had switched to organic produce, but had been unable to find a supplier of organic meats. Now we made it a priority. The morning after Sarah ate organic chicken, her fasting level was again in the normal range. Within the next week, many of her other physical symptoms began to dissipate.

To show that this change was unlikely due to chance, I drew on my background in health economics and analyzed the difference in the fasting glucose levels before and after the change in diet. The results were statistically significant with 99 percent degree of confidence.

Being aware of Sarah's reactions, I became more involved in environmental issues and began to question her and other children's exposures to toxins in different settings such as schools. In order to convince school administrators that their maintenance practices are often harmful to children, you have to have data (although they do occasionally listen to a group of irate parents).

Unfortunately, material safety data sheets are largely incomplete, and the results on long-term reactions of the nervous system and immune system are rarely included. A primary need is more research on long-term health effects, with special emphasis on the variation of symptoms in these children. These data would provide more accurate labeling information as well as serve as a basis for indoor air standards.

When, out of necessity, an individual starts investigating such school products as industrial cleaners, paints, carpets, glues, and roofing materials, the project can take years because of the dearth of information. However, someone must make this effort because school systems and county departments usually purchase through low-bid contracts with scant information on health effects. In their defense, this is partly because they do not have the resources to investigate the products more fully. It is largely a role that only the federal government can fulfill efficiently.

Children with learning disabilities, asthma, and other chronic illnesses breathe neurotoxic gases all day long. Educators wonder why test scores are slipping. It logically follows when you learn about the chemicals used, the lack of fresh air, especially in the winter, and school maintenance done while children are in school. Through passage of a strong Indoor Air Act, we could eventually set meaningful standards that would severely limit the multiple environmental chemicals that assault our children daily.

In the meantime, parents and school personnel need a major education campaign to enlighten them to these issues. Parents assume that the interests of their children are being upheld. How many parents ask principals about the school's pesticide management system or the chemical base of the mopoline used? How many principals and teachers ask the maintenance workers what they are using? Their complacency stems from the assumption that the school system would not be allowed to use materials detrimental to the children or themselves.

To ensure our children's health, the federal government must authorize more research on toxins, especially on the long-term health effects of toxins on the nervous and immune systems of children, improve labeling, pass the Indoor Air Act, and mount a major campaign for parents and school administrators.

Thank you for this opportunity to speak here.

[Prepared statement of Nancy Greenspan follows:]

PREPARED STATEMENT OF NANCY GREENSPAN, PARENT, BETHESDA, MD

My name is Nancy Greenspan. I am here as the mother of three children, one of whom has a chronic illness, and also as the co-founder and director of a local environmental group.

Last year, in recognition of research on the growing link between environmental toxins and chronic illness, the National Academy of Science organized a panel to review this subject. It is one that I have been dealing with since my daughter Sarah was diagnosed with juvenile diabetes at the age of two. For the last six years, my husband and I have been carefully monitoring Sarah's glucose levels, food intake and general environment. Using this information, we have learned that her reaction to chemicals has a dramatic impact on the control of her diabetes. We discovered that petrochemicals, solvents and commercially produced meats (those containing large amounts of growth hormones and antibiotics) profoundly influence her glucose levels and her physical comfort. An aid in our separating out these effects was Sarah's lack of need of insult in her first year after diagnosis. Three short examples illustrate the effects of chemicals on Sarah.

Our first experience with chemical reactions occurred on three consecutive Tuesday evenings when Sarah suddenly had high glucose levels after dinner - an average of 175 rather than the usual 85 to 100. During each of these weeks, these high levels gradually subsided so that by Sunday she was again in the normal range. On the Monday morning of the fourth week I came home to find the cleaning lady mopping the kitchen floor with water and ammonia, a product we had removed from the house because of a general concern with household chemicals. With Sarah starting nursery school, the cleaning lady thought it was safe to use ammonia again. Eliminating the ammonia - and no other change- broke the pattern and Sarah's glucose levels returned to normal.

Over time, as other sudden and mysterious reactions developed, chemicals always seemed to be a factor. We were able to resolve one episode of fluctuating glucose levels by removing natural gas from our house. Whenever Sarah spent any time in one particular room, her levels were higher. On a hunch we had the gas company come and check the pipes. In the part of the basement just below this room, the repair man found a leak. While removing the gas lines from the house, Sarah and I moved in with an understanding neighbor whose house did not contain any gas pipes and Sarah was fine.

Although Sarah's reactions are mostly confined to inhalants - with strong reactions to substances such as pesticides or industrial cleaners - she can have similar reactions to foods containing chemical additives. About six months after we finally started Sarah on insulin - she was 3 1/2 at this time - she fell into a pattern of sleep problems, lethargy, and chronic hunger. Equally troubling, her glucose fasting level doubled. Poring over our records, we noted that her fasting levels were higher on those mornings following nighttime snacks of meat protein.

Months before, we had switched to organic produce, but had been unable to find a supplier of organic meats. Now we made it a priority. The morning after Sarah ate organic chicken, her fasting level was again in the normal range. Within the next week, many of her other physical symptoms began to dissipate. To show that this change was unlikely due to chance, I drew on my background in health economics and analyzed the difference in fasting glucose levels "before and after" the change in diet. The results were statistically significant with 99 percent degree of confidence.

Being aware of Sarah's reactions, I became more involved in environmental issues and began to question her and other children's exposure to toxins in different settings. In order to convince school administrators that their maintenance practices are often harmful to children, you have to have data (although they do occasionally listen to a group of irate parents). Unfortunately, Material Safety Data Sheets are largely incomplete and the results on long-term reactions of the nervous system and immune systems are rarely included. A primary need is more research on long term health effects, with special emphasis on the variation of symptoms. These data would provide more accurate labelling information as well as serve as a basis for indoor air standards.

When, out of necessity, an individual starts investigating such school products as industrial cleaners, paints, carpet glues, and roofing materials, the project can take years because of the dearth of information. However, someone must make this effort because school systems and county departments usually purchases through low bid contracts with scant information on health effects. In their defense, this is partly because they do not have the resources to investigate the products more fully. It is largely a role that only the federal government can fulfil efficiently.

Children with learning disabilities, asthma and other chronic illnesses breathe neurotoxic gasses all day long. Educators wonder why test scores are slipping. It logically follows when you learn about the chemicals used, the lack of fresh air, especially in the winter, and the school maintenance done while children are in school. Through passage of a strong Indoor Air Act, we could eventually set meaningful standards that would severely limit the multiple environmental chemicals that assault our children daily.

In the meantime, parents and school personnel need a major education campaign to enlighten them to these issues. Parents assume that the interests of their children are being upheld. How many parents ask principals about the school's pest management system or the chemical base of the mopoline used? How many principals and teachers ask the maintenance workers what they are using? Their complacency stems from the assumption that the school system would not be "allowed" to use materials detrimental to the children or themselves.

To ensure our children's health, the federal government must

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authorize more research on toxins, especially on the long-term health effects of toxins on the nervous and immune systems of children, improve labelling, pass the Indoor Air Act, and mount a major education campaign for parents and school administrators.

Chairman MILLER. Thank you.
Dr. Schaefer.

STATEMENT OF MARK E. SCHAEFER, Ph.D., PROJECT DIRECTOR, OFFICE OF TECHNOLOGY ASSESSMENT (OTA), U.S. CONGRESS; ACCOMPANIED BY ROGER HERDMAN, M.D., ASSISTANT DIRECTOR, OFFICE OF TECHNOLOGY ASSESSMENT, U.S. CONGRESS

Dr. SCHAEFER. Thank you, Mr. Chairman.

I was formerly a project director at the Office of Technology Assessment, and with me today is Dr. Roger Herdman. He is currently assistant director of OTA, and he is here as the official representative of the agency.

I appreciate being invited to testify on behalf of OTA on the vulnerability of children to neurotoxic substances. This past spring, OTA completed its study entitled "Neurotoxicity: Identifying and Controlling Poisons of the Nervous System." Our report on neurotoxicity is the first of a series of OTA studies on the noncancer health risks posed by toxic chemicals.

More than one out of every five Americans suffers from disorders or disabilities that involve the brain. No one knows precisely to what extent toxic substances contribute to nervous system disorders and disabilities, but they clearly play a significant role, and there is cause for concern.

Neurotoxic substances are chemicals that adversely affect the structure or the function of the nervous system. The nervous system includes the brain, the spinal cord, and the vast array of nerves and sensory organs that control major body functions. Every organ system may be adversely affected by toxic substances, but the nervous system is particularly vulnerable.

Furthermore, the developing nervous system of the fetus and the child is particularly susceptible to these substances. If the damage by a foreign chemical is severe enough to kill a nerve cell, the damage is likely to be permanent, because, unlike many other cells of the human body, nerve cells normally do not regenerate.

At the completion of the developmental process, the brain will be made up of between 10 billion and 100 billion cells, which will make trillions of connections with each other. How the brain correctly "wires" itself is not understood, but the chemical environment of the developing cells is certainly critical to the process.

When the developing nervous system is exposed to neurotoxic substances, the process by which cells multiply and connect may be disrupted, leading, for example, to the severe retardation seen in children exposed to high levels of mercury, perhaps to more subtle deficiencies related to learning and memory, or perhaps to neurological disorders.

Children are exposed to a wide array of chemicals in the environment, including metals such as lead and mercury, pesticides, and a broad range of other pollutants in our air and water and in the food we eat. In recent decades, we have come to rely heavily on chemicals to improve our quality of life. However, our knowledge of the health risks of the substances we are exposing ourselves and our children to has not kept pace with our ambitious efforts to market new and better chemicals.

Few chemicals in commerce have undergone sufficient toxicological testing; therefore, we have an incomplete understanding of the risks most chemicals pose to humans and the environment. Every time we introduce a new chemical into commerce that has not been adequately tested, we take a chance; we gamble with the public's health.

OTA's report describes the state of the present federal regulatory and research system with respect to neurotoxicity. In the course of our study, we found that federal research and testing programs are of insufficient size and scope to address the problems created by neurotoxic substances. Furthermore, little neurotoxicological research is devoted to vulnerable segments of our population, including children.

In total, the national research effort related to developmental neurotoxicology is very small in comparison to the magnitude of the problem. Some agencies place a higher priority on regulatory concerns with respect to neurotoxicity than others. The Environmental Protection Agency, for example, is actively developing and refining developmental neurotoxicology test guidelines that can be used in regulatory programs.

On the other hand, the Food and Drug Administration has the authority to require testing for developmental neurotoxicity but has rarely done so and has no requirement for routine developmental neurotoxicity evaluation. Finally, OTA also found that there is a shortage of adequately trained research and health care professionals to address this problem.

I will only summarize, very briefly, some of the steps Congress might consider if it wishes to take further action to address this problem. From the regulatory standpoint, numerous laws either directly or indirectly control neurotoxic substances. Congress could mandate more extensive neurotoxicity testing under the Toxic Substances Control Act or the Federal Insecticide, Fungicide, and Rodenticide Act, and it could mandate that neurotoxicity concerns be given greater attention when making regulatory decisions with respect to toxic substances.

In regard to federal research efforts, Congress could take steps to enhance neurotoxicology programs at various agencies. With respect to the research and health care personnel issue, Congress could take steps to enhance pre- and post-doctoral research training programs in neurotoxicology.

Finally, assuring that the public is adequately informed of the risks that toxic substances pose to themselves and their children is of major importance. Congress could take action to ensure that workers, particularly women of child-bearing age, receive sufficient information on the neurotoxic potential of chemicals to which they are exposed, both at home and in the working environment.

In addition, Congress could require that neurotoxicity concerns be explicitly described in information developed and released under the Federal Emergency Planning and Community Right-to-Know Act, and it could mandate improved labeling of consumer products with respect to potential neurotoxic effects.

Mr. Chairman, thank you again for inviting OTA to testify today on this important issue. We would be happy to assist the committee in the future as your work in this area progresses.

[Prepared statement of Mark E. Schaefer, Ph.D., follows:]

PREPARED STATEMENT OF MARK E. SCHAEFER, PH.D., PROJECT DIRECTOR, OFFICE OF TECHNOLOGY ASSESSMENT, U.S. CONGRESS

VULNERABILITY OF CHILDREN TO NEUROTOXIC SUBSTANCES

Mr. Chairman, I am Mark Schaefer, I was formerly a Project Director at the Office of Technology Assessment. With me is Dr. Roger Herdman, currently Assistant Director of OTA, as official representative of the agency. I appreciate being invited to testify on behalf of OTA on the vulnerability of children to neurotoxic substances. This past Spring, OTA completed its study entitled "Neurotoxicity: Identifying and Controlling Poisons of the Nervous System." The report is the product of the collective efforts of a number of OTA staff and outside consultants who are listed at the beginning of the document, and I would like to acknowledge their excellent contributions. Our report on neurotoxicity is the first of a series of OTA studies on the noncancer health risks posed by toxic chemicals.

As you are aware, the President designated the 1990s as the Decade of the Brain following the passage of a Joint Resolution by Congress in the Summer of 1989. The first paragraph of the Resolution includes an ominous statistic:

> Whereas it is estimated that 50 million Americans are affected each year by disorders and disabilities that involve the brain, including the major mental illnesses; inherited and degenerative diseases; stroke; epilepsy; addictive disorders; injury resulting from prenatal events, environmental neurotoxins, and trauma; and speech language, hearing and other cognitive disorders. (Public Law 101-58)

In other words, more than one out of every five Americans suffers from disorders or disabilities that involve the brain. No one knows precisely to what extent toxic substances contribute to nervous system disorders and disabilities, but they clearly play a significant role, and there is cause for concern. As you know, lead poisoning alone is a very serious public health problem that threatens the intellectual capabilities of a large number of the nation's children.

Public concern about exposure to toxic substances tends to focus on whether or not a chemical might cause cancer. Noncancer health risks including adverse effects on organs and organ systems -- the nervous system, the kidney, the liver, the heart, the immune system -- are of comparatively little concern. Cancer is a very serious health problem, but we should not let our fear of this disease blind us to the many other potentially serious consequences of exposure to toxic substances.

Neurotoxic substances are chemicals that adversely affect the structure or function of the nervous system. The nervous system includes the brain, the spinal cord, and the vast array of nerves and sensory organs that control major body functions. Movement, thought, vision, hearing, speech, heart function, respiration, and numerous other physiological processes are controlled by this complex network of nerve processes, transmitters, hormones, receptors, and channels.

Every organ system may be adversely affected by toxic substances, but the nervous system is particularly vulnerable. Furthermore, the developing nervous system of the fetus and the child is particularly susceptible to these substances. It therefore makes sense for policy-makers concerned about the noncancer health risks posed by toxic substances to focus particular attention on the nervous system of the child. First, I would like to briefly explain why the developing nervous system is vulnerable to toxic substances, then I will describe some of the findings of our study and possible policy approaches to better address this important public health problem.

There are several reasons why the nervous system is particularly susceptible to toxic substances. The brain, spinal cord, and nerves throughout the body depend on a delicate balance of chemicals for proper functioning. There are many opportunities for foreign chemicals to disrupt this balance. For example, they may block the channels through which charged sodium or potassium molecules flow and thereby disrupt electrical signalling in cells. Toxic chemicals may disrupt the synthesis or degradation of neurotransmitters, the chemical messengers that travel between nerve cells. They may block receptors, the sites at which neurotransmitters attach in a lock and key fashion to affect chemical changes in other nerve cells. Toxic chemicals can also disrupt the activity of the enzymes that catalyze the biochemical reactions that take place within nerve cells. And while most cells in the human body are very small, nerve cells can have long processes, providing a vast surface area for chemical attack. If the damage caused by a foreign chemical is severe enough to kill a nerve cell, the damage is likely to be permanent, because unlike many other cells of the human body, nerve cells normally do not regenerate.

It is useful to examine the fundamental steps in nervous system development in order to understand why exposure to toxic chemicals during this period is so dangerous. Early in fetal development a flat sheet of about 125,000 cells forms which rolls into a tube, called the neural tube. In the following weeks and months, the cells of this tube multiply, migrate, and begin differentiating into the specific cell types of the nervous system. The neurons begin to extend processes called axons and dendrites which link up with other cells into an exquisite network of very precise and highly complex connections. At the completion of the developmental process the brain will be made up of between 10 billion and 100 billion cells which make trillions of connections with each other. How the brain correctly "wires" itself is not understood, but the chemical environment of the developing cells is certainly critical to the process. When the developing nervous system is exposed to neurotoxic substances, the process by which cells multiply and connect may be disrupted, leading, for example, to the severe retardation seen in children exposed to high levels of mercury, or perhaps to more subtle deficiencies related to learning and memory, or to neurological disorders.

In the fully developed nervous system, the brain and spinal cord are partially protected from some toxic substances by a layer of tightly juxtaposed cells in blood vessel walls know as the blood-brain barrier. This barrier functions as a selective filter allowing some compounds to pass through while keeping others out. The fetal brain with its incompletely developed blood-brain barrier can be attacked by toxic substances that might have little effect on the mother's brain.

Finally, developing organs are less well equipped to detoxify foreign substances. The liver is the principal organ involved in detoxification, but nearly all tissues, including those of the nervous system, have some capacity to detoxify chemicals. Since the detoxification systems of developing tissues are not fully functional, foreign chemicals can more readily adversely affect biochemical and physiological systems.

Children are exposed to a vast array of chemicals in the environment, including metals such as lead and mercury, pesticides, and a broad range of other pollutants in our air and water and in the food we eat. In recent decades, we have come to rely heavily on chemicals to improve our quality of life. However, our knowledge of the health risks of the chemicals we are exposing ourselves and our children to has not kept pace with our ambitious efforts to market new and better chemicals. There are already more than 65,000 chemicals in the U.S. Environmental Protection Agency's inventory of toxic substances and each year the Agency receives some 1,500 notices of intent to manufacture new substances. Although the vast majority of these chemicals are probably harmless at low levels of exposure, some are not, and the health threats they pose should be taken seriously. The difficulty we face is that few chemicals in commerce have undergone sufficient toxicological testing. Therefore, we have an incomplete understanding of the risk most chemicals pose to humans and the environment. Every time we introduce a new chemical into commerce that has not been adequately tested, we take a chance and gamble with the public health.

One class of chemicals, the organophosphate and carbamate insecticides, are all neurotoxic to varying degrees. Indeed they are effective in killing insects because of their neurotoxicological and other properties. The difficulty is that at the molecular and cellular levels, the nervous systems of insects and humans are similar in many respects; consequently, insecticides can adversely affect humans as well. The reason insecticides are normally not harmful to us is that the dose to which we are exposed is comparatively very small. However, no one is sure what level of insecticide exposure is safe for a given individual. As you recall, last year the Natural Resources Defense Council released a study which raised concerns that children are being adversely affected by pesticide residues on fruit and vegetables.

OTA's report describes the state of the present federal regulatory and research system with respect to neurotoxicity. In the course of our study, we found that federal research and testing programs are of insufficient size and scope to address the problems created by neurotoxic substances. Little neurotoxicological research is devoted to vulnerable segments of our population, including children. The Food and Drug Administration's National Center for Toxicological Research has a small research program devoted to developmental neurotoxicology, and projects are underway at other federal laboratories. Also, a number of investigators in academia are being supported by grants from the National Institute of Environmental Health Sciences. However, in total, the national research effort related to developmental neurotoxicology is small in comparison to the magnitude of the problem.

Some agencies place a higher priority on regulatory concerns with respect to neurotoxicity than others. The Environmental Protection Agency (EPA), for example, is actively developing and refining developmental neurotoxicology test guidelines that can be used in regulatory programs. Recently, EPA and the National Institute on Drug Abuse (NIDA) sponsored a workshop to examine a range of issues associated with the use of developmental neurotoxicity testing data in regulatory programs. The Food and Drug Administration has the authority to require testing for developmental neurotoxicity but has rarely done so and has no requirement for routine developmental neurotoxicity evaluations.

OTA also found that there is a shortage of adequately trained research and health-care professionals to address the neurotoxicity problem. Improving the federal response will require funds to train more scientists to conduct research in this area and to train physicians, nurses, and others to recognize and treat the adverse health effects caused by exposure to neurotoxic substances.

The OTA report on Neurotoxicity describes policy issues and options for congressional action in six broad categories: 1) adequacy of the Federal regulatory framework, 2) adequacy of Federal and federally sponsored research programs, 3) coordination of Federal regulatory and research programs, 4) availability of adequately trained research and health-care professionals, 5) communication of information to workers and the public, and 6) adequacy of international regulatory and research programs.

I will only summarize very briefly some of the steps Congress could take if it wished to take further action to address the neurotoxicity problem with respect to children. From the regulatory standpoint, numerous laws either directly or indirectly control neurotoxic substances. Congress could mandate more extensive neurotoxicity testing under the Toxic Substances Control Act (TSCA) or the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA); it could mandate that neurotoxicity concerns be given greater attention when making regulatory decisions with respect to toxic substances; and it could enhance federal laws focusing specifically on exposure of children to lead.

With respect to federal research efforts, Congress could take steps to enhance neurotoxicology programs at or sponsored by the Environmental Protection Agency; the National Institutes of Health; the Alcohol, Drug Abuse, and Mental Health Administration; the Food and Drug Administration; the National Institute for Occupational Safety and Health; and other agencies. Various specific possible actions are described in our report.

With respect to the research and health care personnel issue, Congress could take steps to enhance pre- and post-doctoral research training programs in neurotoxicology by providing funds to support more training grants to individuals and/or research centers.

Finally, assuring that the public is adequately informed of the risks that toxic substances pose to themselves and their children is of major importance. Congress could take action to ensure that workers, particularly women of child-bearing age, receive sufficient information on the neurotoxic potential of chemicals to which they are exposed, both at home and in the working environment. In addition, Congress could require that neurotoxicity concerns be explicitly described in information developed and released under the Federal Emergency Planning and Community Right-to-Know Act, and it could mandate improved labeling of consumer products with respect to potential neurotoxic effects.

Mr. Chairman, thank you again for inviting OTA to testify today on this important issue. We would be happy to assist the Committee in the future as your work in this area progresses.

Chairman Miller. Thank you.

Dr. Needleman.

STATEMENT OF HERBERT L. NEEDLEMAN, M.D., PROFESSOR OF PSYCHIATRY AND PEDIATRICS, UNIVERSITY OF PITTSBURGH, SCHOOL OF MEDICINE; CHAIRMAN OF THE ALLIANCE TO END CHILDHOOD LEAD POISONING, PITTSBURGH, PA

Dr. Needleman. Good morning, Mr. Chairman.

I am Herbert Needleman. I am professor of psychiatry and pediatrics at the University of Pittsburgh. I am a member of the Committee on Environmental Hazards of the American Academy of Pediatrics, and the Institute of Medicine. I am also the chairman of a new organization, the Alliance to End Childhood Lead Poisoning. I am very happy to be here.

I have submitted, in addition to my testimony, two recent publications of my group that deal with the long-term effects of lead poisoning: The first is from the New England Journal of Medicine, and the second is from the Journal of the American Medical Association. That paper which is a meta-analysis, a quantitative review of all modern studies of low-level lead exposure in children.

I can summarize my testimony by making three points: The first is that lead exposure remains perhaps the most serious American pediatric problem. Mr. Sikorski quoted the ATSDR report, which says that three to four million children have levels of lead over 15 micrograms per deciliter, the current, widely-accepted threshold for toxicity.

One child in six, regardless of race or class—exceed that threshold. Half of poor, black children start school with toxic levels of lead above 15 microgram per deciliter in their blood.

Second: Lead poisoning is completely preventable. It is an eradicable disease. We should be aiming not to control it but to wipe it out. In fact, the government is beginning to pay serious attention to that possibility. Assistant Secretary Mason has directed CDC to prepare a strategic plan to eliminate lead poisoning, to eradicate it over the next 15 or 20 years. That plan is on his desk.

The third point is that in eradicating lead poisoning we have an opportunity to accomplish an enormous number of important social goods, like dealing with unemployment and returning housing to decent circumstances.

Lead affects many targets in the body—children are more susceptible—but the most important is the child's brain. It is now unarguable that levels of lead that do not display clinical symptoms are associated with lower IQ points, attention disorders, and behavioral disturbances.

My group has contributed to that data base. In 1979, we reported that children in Boston area school systems who had no symptoms of lead poisoning but had higher levels of lead in their teeth had lower IQ scores, more disorder behavior in class, and poor attention.

We have followed those subjects up. They are now adults, and, in January of 1990, we reported in the New England Journal of Medicine that having high lead in your teeth in 1976 was associated with a sevenfold risk for failing to graduate from high school, a six-fold risk for reading disabilities, and a lower class standing in the final year of high school, more absenteeism in the final year of high school, lower vocabulary scores, disturbances in fine motor function.

So the news is that low-level lead exposure produces permanent defects; defects that are not just numbers on an IQ score but have to do with how much money one would make and where one will be placed in society.

Lead crosses the placenta. You can measure it in the umbilical cord blood. David Bellinger, Alan Levitton, and I studied 5,000 births at the Boston Hospital for Women, and showed that having a level of lead in the umbilical cord blood over 10 micrograms per deciliter was associated with lower IQ scores at two years of age. That has been replicated in Australia and in Cincinnati, so there is good convergence on the effects of lead on the fetus in utero.

I want to spend one minute on the fact that the difference between exposed and unexposed children at these doses, the mean difference is about four to six points in the IQ, but shifting that distribution quadruples the rate for severe deficit. It drives the proportion of children with IQ scores below 80 from 4 percent to 16 percent.

In addition to the 400 percent increase in severe deficit, there also is a shift at the top end of the curve. Not every child who had high lead in his or her teeth was dull. The highest IQ score we discovered was 125, but five percent of the children who had lower levels exceeded that, as high as 143. One of costs of low-level lead exposure is that it prevents five percent of our population from achieving superior function.

That is a social disaster for this country. That means that perhaps two million American children are not achieving superior function. The social cost of that would be extraordinarily high.

We have shown, and others have as well, that lead is associated with attention deficit and hyperactivity. Teachers reliably reported a dose-related rate of increase in hyperactivity, and hyperactivity is a strong risk factor for delinquency and criminal behavior. A high proportion of criminals have a history of hyperactivity.

We can estimate an attributable risk for delinquency given elevated lead. The attributable risk for hyperactivity given lead is .5. Half of the children with elevated lead in their teeth are hyperactive. If you have hyperactivity, the attributable risk for delinquency is about .4, so the joint probability is about .2.

That permits us to estimate the lower bound for attributable risk for delinquency given elevated lead at 20 percent, and we are studying that at this time. It is a reasonable hypothesis that a measurable proportion of delinquent children have that disturbance due to their early lead exposure.

Why, in fact, has so little attention been given to eradicating this disease? This is not, after all, molecular biology. It is easy to measure lead on the walls; it is easy to get rid of. There are at least five reasons: The first is that for a long time it has been assumed to occur only among poor, inner-city minorities. Somehow that has gotten perverted to mean that the mother's rearing style is responsible for the child's lead exposure: If the mother had taken care of the child, this wouldn't have happened. Once that happens, the responsibility of government or other organizations to deal with it is dismissed.

The second is that since the Lead Paint Poisoning Prevention Act of 1972 was passed, and since lead has been taken out of gasoline recently, many people, including pediatricians, believe that the disease is gone. Of course, passing the act did nothing to take lead paint off the walls of two million American houses. There are two million American houses which have deteriorated leaded surface in which children live, and that is an obscene circumstance.

The third is that the lead industry has spent a great deal of money and rented scientists to try to obscure the relationship between lead exposure and deficit.

The fourth is that this is not a very dramatic, high-tech disease, and the academic culture does not find it fascinating. There are many good institutions, pediatric teaching hospitals, in which screening for lead no longer continues to be practiced.

The fifth is that certain segments of the government, have been derelict in attending to this.

I will finish by suggesting that if we had a computer program and could map where lead is in this country in superabundance, we could print that out. Then if we changed the program to count where jobs are in short supply, we could print that map. Then if we also asked where decent housing was in short supply, we would have three maps and they would be virtually identical.

You have the simultaneous superabundance of lead, shortage of jobs and housing. What would we do, if we weren't bound by conventions, about that disequilibrium? Well, it might make sense to train the unemployed in safe de-leading, and pay them for it, and for the same dollar we could reduce unemployment, return houses to circulation, and eradicate lead poisoning.

That is a prospect that is very exciting to me. That is why we formed the Alliance to End Childhood Lead Poisoning, and that will be our project for the next two or three years.

Thank you.

[Prepared statement of Herbert L. Needleman, M.D., follows:]

Prepared Statement of Herbert L. Needleman M.D., Professor of Psychiatry and Pediatrics, University of Pittsburgh, School of Medicine, Chairman, the Alliance To End Childhood Lead Poisoning, Pittsburgh, PA

Good morning Mr. Chairman. I am Herbert L. Needleman M.D., professor of psychiatry and pediatrics at the University of Pittsburgh. I am chairman of the Alliance to End Childhood Lead Poisoning. I am also a member of the Committee on Environmental Hazards of the American Academy of Pediatrics, and the Insttute of Medicine of the National Academy of Sciences. I welcome this opportunity to appear before this committee to discuss the impact of lead at low dose on the welfare of children. Lead poisoning is a subject I and my colleagues have been investigating, with support from the federal government, for 20 years. I submit for the record two publications of mine published in January and February of this year on this subject. One is from the *New England Journal of Medicine*, and reports the long term effects, in adulthood, of exposure to lead at low dose as a child. The second is from the *Journal of the American Medical Association*, and is a quantitative review of all the published modern studies of lead at low dose.

I want to make the following points: 1) Lead poisoning is the most serious pediatric health problem in the United States today; 2) Lead poisoning is completely preventable; 3) In eliminating this disease, we can also attack other fundamental problems of poverty.

Lead toxicity, like most of the serious threats faced by this planet -- and this Committee -- is a product of human activity and choice. Our understanding of the nature of the disease, its origins, and the steps to its remedy has grown rapidly in the past 5 years. But effective prevention, with two exceptions, has been feeble and halting. The defined threshold for toxicity in children, once considered to occur at 40µg/dl, has been reset at 10-15µg/dl by the Federal Government on the basis of the latest science. This means that 3-4 million American children are at risk for central nervous system damage. Exposure to lead is the most serious disease of childhood in the United States. It has recently been recognized as a serious problem in Europe, Scandinavia and Australia.

Newer studies of the biology of lead exposure have demonstrated effects in systems heretofore not known to be vulnerable, at doses heretofore thought to be harmless. Epidemiological studies of children using larger samples, better covariate identification and control, more sophisticated statistical modelling have shown deficits at lower levels of burden. The no-effect level has not been found. Quantitative reviews, or meta-analysis, of the literature of low dose IQ effects have shown a striking convergence of scientific opinion on the reality of low dose damage. The question of whether low level effects damage children are real is now only raised by the uninformed or by those with economic interests that would be damaged by true control of the toxicant.

I want to briefly review what we know about low level lead exposure and children's IQ's. There are 24 modern studies of this relationship in the literature. Because of the limitations of time, I will concentrate on the work of my group, begun when I was at Harvard Medical School. We used, for the first time in outcome studies, dentine lead as the exposure marker; we measured IQ and other behaviors by a number of sensitive tests; we controlled for 39 factors that could confound; and we selected our sample in an unbiased fashion. We found that having more lead in one's teeth was associated with lower IQ, speech and language handicaps, and poor attention. Teachers, blind to the children's lead levels, reported that as tooth lead went up, bad classroom behavior became more common. This study was published in the _New England Journal of Medicine_ in 1979 at the time the EPA was struggling with writing an air lead standard, and was used by the administrator in reducing lead in the atmosphere and in gasoline.

We then went on to design and execute the first study of lead exposure during pregnancy and its effects on infant development. We studied umbilical cord blood leads and outcome in 5000 births and found that in our sample, lead did not affect birth weight, but was related to the rate of minor (non threatening) malformations. Minor malformations are of little consequence in and of themselves, but are predictive of undiagnosed major malformations and later behavioral aberration.

We have followed 249 of these infants at 1, 6, 12, 24, 48, and 57 months of age, and found that prenatal exposure to lead was related to mental development as late as 24 months of age. At 57 months, the effect of postnatal exposure to lead became dominant, although the prenatal effect was still measurable in the children of low socioeconomic status. These children continue to be followed, and we hope to be able to track them into their adolescence. The high lead group had blood lead levels above 10 µg/dl. There are 400,000 children born each year in that range.

We have followed the subjects from our tooth study into adulthood, to find results that are even more disturbing. This was reported in the New England Journal of Medicine in January. Having high lead in one's teeth carried a seven-fold increase in the risk of nongraduation from high school, and a six-fold increase in the risk for reading disability. The social costs for this exposure are staggering. Lead is one of the preventable causes of school failure and reading disabilities. In addition, we have shown that small shifts of the distribution in IQ scores in relation to lead exposure quadruple the risk for severe deficit (Scores below 80). They also reduce the number of children at the top end of the distribution. This means that the number of children with superior IQ'S (over 125) will be reduced by 50%. In a population of 200 million, this means about 2 million people will be deprived of achieving this level of competence. This is a personal and national disaster.

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Studies of lead exposure have focused on IQ. There is no good reason for this: more critical changes in the brain can be expressed in other behaviors. We and others have shown that lead is associated with attention deficits and hyperactivity. This collection of behaviors has dire implications for adjustment to society. If a male child has hyperactivity and one sign of conduct disorder, he has a 60% chance of appearing on a police blotter multiple times before the age of 18. Wilson and Herrnstein report that criminality is constitutional in nature, and cite the following findings in support of this claim: criminality can be diagnosed early in childhood; it is more common in males; it is more common in blacks; it is more common in urban areas; criminals have lower IQ's; possess a history of hyperactivity; and come from disorganized homes. All of these are risk factors or effects of lead exposure. This is not to say that lead is responsible for all crime; life is more complex than that. But it is a reasonable hypothesis that some of the disordered behavior of criminals is a function of disordered brain function, and that some of this derives from lead.

On the basis of these studies and many similar reports from the U.S., Europe and Australia, the Agency for Toxic Substances and Disease Registry, in its recent Report to Congress, has concluded that neurotoxicity from lead begins at blood lead levels as low as 10-15 µg/dl. This means that one American child in six has toxic amounts of lead in his or her blood, and that 400,000 newborns are delivered bearing toxic levels each year. Lead is not a problem for poor inner city minorities alone. But like many of the assaults upon decent living, the poor receive a unfair dose. For black children in poverty, the rate of blood leads over 10µg/dl is 55%!. This datum, one of the most outrageous and frightening public health statistics, has received only passing attention since the ATSDR report. It means that lead exposure is among the most serious American public health problems.

One is forced to ask why lead poisoning, long known, not nearly as complex as AIDS or cancer, has not been remedied. There are four reasons: First, it is generally believed that lead poisoning is a disease of the poor, and that inferior child care is at the root. Once the victim has been blamed, the public and professional consciences can rest. Second is that removing lead from gasoline, from water, and from housing costs money, and this results in the exertion of vested interests, who know how to use the strings of power. The lead industry has worked mightily to obscure the effects of lead at low dose. It has often used individuals from the academic world in this effort. In 1933, almost 60 years ago, the Lead Industries Association exerted pressure to alter regulations that would have limited the use of white lead in building paint. Third, lead is a low technology disease, it does not enjoy the cachet of lasers, molecular biology or liver transplants. It is not at the center of the medical drama, and many pediatricians believe that with the removal of lead from gasoline, and the passage of the lead paint act, the problem has been solved. Many academic pediatric centers have stopped testing for lead, and many pediatric trainees no longer consider it in making a differential diagnosis of developmental failure. Finally, government has been slow to realize the dimensions of the problem and deal with it appropriately. Egregious in this respect has been HUD, who have failed utterly to protect residents in HUD owned or supervised property from lead poisoning.

There is a striking disequilibrium in the distribution of risk. If one were to map the areas where lead is to be found in excess, and then map where decent housing was in short supply, and finally map where decent jobs were scarce, the three maps would be isomorphic. What could be done to rationalize this imbalance? One simple solution would be to train unemployed people from high lead areas in safe deleading and housing rehabilitation, and to pay them a living wage. This would reduce unemployment, make more decent dwellings available, and reduce lead exposure for children. This is an expensive enterprise. There are 2 million homes in the

U.S. which are deteriorated, have leaded surfaces, in which children live. These are the pest houses of the 20th century. It costs $5000 to delead a small home. That means $10 billion would be required for this project. For $6 billion a comprehensive employment and training program for 40,000 unemployed citizens paying them $15,000 per year for 10 years could be funded. This would leave $4 billion for the training cadre, supplies, insurance and administrative costs. Some of this money would be returned in taxes; almost all would circulate in the inner city and multiply by creating demand for on groceries, laundry, and other goods and services.

This may strike some as a utopian phantasy. It is instructive to realize that the cost of constructing one new prison bed is $30,000 and maintaining one prison inmate is $20,000-30,000 per year. Pay now or pay later. A recent front page story in the New York Times told of major industrialists deeply troubled because they are unable to find qualified employees to operate the factories of today. Modern industrial workers increasingly need to be literate, and competent in mathematics and problem solving. Management is having difficulty recruiting workers who meet the present day criteria, and project that if this isn't remedied, we will become less and less competitive. We risk becoming an underdeveloped nation. We have seen persuasive evidence that one of the identifiable causes of school failure and reading disabilities is lead.

Lead poisoning is no mystery; the toxin's presence, its effects, and the steps to removing it forever are plainly prescribed. What is needed is the same kind of vision displayed by the people from DHSS who set out to eliminate smallpox from the earth. They were greeted with skepticism when they first proposed this effort. Smallpox is now a disease of historical interest. This government, given the same degree of vision and committment, can in a decade be credited with having wiped out this terrible, ubiquitous, silent destroyer of our children's brains and futures.

Chairman MILLER. Thank you.
Mr. Wilson.

STATEMENT OF RICHARD WILSON, MALLINCKRODT PROFESSOR OF PHYSICS, HARVARD UNIVERSITY, CAMBRIDGE, MA

Mr. WILSON. Mr. Chairman, congressmen; ladies and gentlemen, I want to explain to you the way in which a professional assessor and analyzer of risks addresses the questions you are concerned with. I would also note, I was born and brought up at a place and time when the blood level averaged 40 micrograms per deciliter, which was quite high, and I am glad it is not as high as that now. I would also note, I am the father of six children, five of whom have food allergies.

In coping with environmental toxins or any other potential hazard, I like to characterize the possible approaches and steps of increasing sophistication: First, an absolute ban; the second, using the best available technology; and the third would be risk assessment, analysis and balancing of risks and benefits.

The taboo of primitive societies was to ban something we did not understand and could do without. Even in modern society, an absolute ban is still often considered. The Delaney clause of the FDA is a good example. When one part of society needs or wants something that another part wants to ban, it has been a practice to reduce the exposure by using the best available control technology to avoid the absolute bankruptcy of an industry rather than using an absolute ban.

We have tended to follow those two procedures. As a result, it is estimated the cost to U.S. industry of reducing exposure to environmental toxins has reached $100 billion a year and is rising fast. I support spending money to reduce environmental and public health issues, but this sum is large enough that it is important that it be wisely spent and spent to improve public health and well-being, particularly of children who are the future of our society.

This demands, in my view, that the third, more sophisticated approach of risk/benefit analysis be used. Over the last 30 years, professionals in health and safety began to adopt a language, that of risk rather than of absolute safety. Although there may be a threshold below which an environmental toxin has no effect, there may not be. Then there is a risk that there is an adverse health effect, and an important issue rises: What is the magnitude of the risk?

Once the language of risk is used, it must also be realized there is no possibility of zero risk. A second question must thus be asked: What magnitude of risks are acceptable? A third question could be: Is there an alternative action with less risk?

Because the very word "risk" implies uncertainty, understanding uncertainty is at the center of understanding risk. Agencies tend to avoid understanding uncertainty by trying to regulate on an upper limit of risk. I believe they cannot do this consistently. Anyone can give an upper limit below which he believes the risk will be zero. The less knowledge he has, the bigger the upper limit. Then the statement often becomes irrelevant and useless.

It is vital that an agency have a procedure for reducing upper limits as the science improves, but I know of no agency which does. Any regulation based on an upper limit of risk may be unnecessarily strict. I prefer one based on scientists' best estimates, perhaps obtained by a method which is becoming increasingly common of solicitation of expert opinion.

It must not be thought that scientists and risk analysts ignore effects of toxins on children's health. On the contrary, risk analysis is an excellent procedure to enable the special sensitivity of children to be explicitly recognized.

For example, in 1928, the International Commission on Radiological Protection recommended regulations which have been adopted by most of the countries of the world. Among the present regulations are special ones to limit occupational radiation exposure to those under 18 and to pregnant women, recognizing it is likely, but not proven, that children and the infant fetus are especially sensitive to radiation.

In the medical profession, there are special recommendations to reduce unnecessary x-rays to children and to pregnant women.

Two years ago, when it was realized that children were particularly exposed to ALAR and even more to its metabolite, UDMH, there was and is scientific disagreement on whether the chemicals are carcinogenic enough for the exposure to cause special risk. Although the EPA administrator was slow to act, I disagreed with him, Mr. Jack Moore, when he stated on "60 Minutes" that the laws did not permit him to act fast.

He could have done one of the following three things: asked for an emergency suspension of registration; two, "jaw-boned" the apple industry; or, three, advised the state commissioners of health to use their emergency powers which exceed that of any federal agency.

I believe that his own hesitation in not using the emergency powers was the knowledge that many scientists on EPA's Science Advisory Board did not agree that the risk was large and certainly not large enough to demand an emergency power. Whatever the laws at his disposal, even if he had new ones, presumably the same scientists would have the same hesitation.

Without taking a position on whether or not ALAR should be banned, I note that the risk of drinking and using water from the usual treated city waters of our major cities, calculated using the same pessimistic, conservative procedure, is 5 or 10 times greater because of chloroform in the city water than that from ALAR. It is likely to be dominated by the use of water for bathing children and absorption of chloroform through the skin. So it is very important for children.

Asbestos provides another example. It is well known that there were large exposures in the workplace 30 years ago that are still producing lung cancers, particularly among smokers. In the environment, exposures are typically a thousand times smaller, and for a long time they were ignored. However, 15 years ago, some risk assessors began to suggest that there may not be a threshold below which there is no risk, and, therefore, the lower environmental exposures pose some risk.

Moreover, it was pointed out that the rare cancer, mesothelioma, which is caused by asbestos, has a long latent period. This means that occupational exposures are too late to allow expression of the disease before death, and use of occupational exposures to predict what children's exposures might do may understate the risk.

This realization, urged particularly by Professor Julian Peto of London, quickly led to suggestions that damaged asbestos in schools might be dangerous and led to demands for strong action. While I personally believe that in most cases it is best to leave the asbestos in place, I note that there is plenty of power for local, state, or federal authorities to order removal of asbestos when they want to.

What is needed is better use of scientific information so that agencies can act in the best way for public health and decide whether the enormous amounts they are called upon to spend are indeed likely to be accompanied by improvements in health.

After the federal government has set a reasonable level of safety, I see no reason why citizens should not decide to go further on their own, at their own expense. For example, the EPA called upon schools to study the effect of asbestos in schools, not necessarily to remove it. It is and should be up to the local people to decide whether to make the expenditures necessary or whether there are other expenditures they can do for the children's welfare and public health.

The federal government should not bias the decision by providing money, nor should they be allowed to bias the decision by allowing someone whose profession is to remove asbestos to make the recommendation.

Our agencies are, by their nature, adversarial. We ask and expect that EPA continually urge the cause of environmental protection. FDA must push for safe food and drugs. On the other hand, we ask and expect that Commerce and DOE work for cheaper and more plentiful goods and energy. Therefore, any balancing of these needs must ultimately be external to these agencies.

For financial matters, it is the Office of Management and Budget, but for scientific matters there is no such balancing now being done. We need similar things for the scientific matters, and I suggest perhaps the Office of Science and Technology Policy in the White House could take a lead in this. Congress may wish to ask for this and provide the budget for it.

With all the toxins facing us, it is important that there be research on which are the most likely to be especially risky to children. Exposure to children is especially important for carcinogens with a long latent period, which are often called early-stage carcinogens. A carcinogen with a short latent period is often a promoter and is usually most important at a later age.

Unfortunately, there is at the moment little scientific consensus about which of the known toxins, let alone which of the known chemicals which might be toxins, are in which category. The upper limit of this must therefore be kept high enough to encompass these uncertainties. If we insist on regulating the upper limit of this, and to a low-risk number, enormous sums can be spent with the likely improvement on public health being zero.

I hope that future science can help elucidate these matters. Thank you for your attention.

[Prepared statement of Richard Wilson follows:]

PREPARED STATEMENT OF RICHARD WILSON, MALLINCKRODT PROFESSOR OF PHYSICS, HARVARD UNIVERSITY, CAMBRIDGE, MA

Congressmen and women; ladies and gentlemen. I am grateful for your attention. I come to explain to you the way in which a professional assessor and analyst of risks addresses the questions with which you are concerned.

In coping with environmental toxins or any other potential hazard, I like to characterize the possible approaches in steps of increasing sophistication.

The absolute ban (or taboo)
The best available technology
Risk assessment, analysis and balancing

The taboo of primitive societies was to ban something we did not understand, and could do without. Even in modern society an absolute ban is still often considered; the Delaney clause of the FDA is a good example. When one part of society needs or wants something that another part wishes to ban, it has been a practice to reduce the exposure by using the Best Available Control Technology rather than using an absolute ban. The best available control technology is, in principle, without regard to expense, but in practice, expense that would bankrupt an industry is avoided.

We have tended to follow there two procedures. As a result, it has been estimated that the cost to U.S. industry of reducing exposure to environmental toxins has reached $100 billion and is rising fast. I support spending money on environmental and public health issues, but this sum is large enough that it is important that it be wisely spent -- and spent to improve public health and wellbeing, particularly of children who are the future of our society. It is this I believe risk analysts should address. This demands that the third, more sophisticated approach, risk/benefit analysis, be used.

Over the last 30 years, professionals in health and safety began to adopt a language, that of risk, rather than of absolute safety. Although there may be a threshold below which an environmental toxin has no adverse effects, there may not be. Then there is a risk that there is an adverse health effect, and an important issue arises: "What is the magnitude of the risk?" Once the language of risk is used it must also be realized that there is no possibility of zero risk. A second issue must then be stated: "What magnitude of risks are acceptable?" A third issue could be: "Is there an alternative action with less risk?"

Laws and regulations have only begun to recognize this new, powerful language and analysis technique. As it is recognized there is also a tendency which we must fight to misuse the language and pervert the analysis technique.

Because the very word "risk" implies uncertainty, understanding uncertainty is at the center of understanding risk. Agencies tend to avoid understanding uncertainty by trying to regulate on an "upper limit" of risk. This they cannot do consistently.

Anyone can give an "upper limit" below which the risk will in his opinion lie; the less knowledge he has the bigger the upper limit. Then the statement often becomes irrelevant and useless. It is vital that an agency have a procedure for reducing upper limits as the science improves. But I know of no agency with such a procedure. Any regulation based on an "upper limit" of risk may be unnecessarily strict; I would prefer one based on scientists' best estimates -- perhaps obtained by the developing method of "solicitations of expert opinion."

It must not be thought that scientists and risk analysts ignore effects of toxins on children's health. On the contrary, risk analysis is an excellent procedure to enable children's sensitivity to be explicitly recognized. I contend that no special laws and regulations are needed to ensure this. I show this by some examples.

Since 1928 the International Commission on Radiological Protection has recommended regulations on radiation to the countries of the world. Among the present regulations are special ones to limit occupational radiation exposure to those under 18 and to pregnant women recognizing that it is likely (although not proven) that children (and the infant fetus) are especially sensitive to radiation. In the medical profession there are special recommendations to reduce unnecessary x-rays to children.

Two years ago, when it was realized that children were particularly exposed to ALAR (daminozide) and even more to its metabolite UDMH, there was, and is, scientific disagreement on whether the chemicals are carcinogenic enough for the exposure to cause special risk. Although the EPA administrator was slow to act, I disagree with Jack Moore, when he stated on "60 Minutes" that the laws did not permit him to act fast. He could have:

1) asked for an emergency cancellation of registration
or 2) "jaw-boned" the apple industry
or 3) Advised the State Commissioners of Health to use their emergency powers.

I believe that his only hesitation in using the emergency powers was the knowledge that many scientists on EPA's Science Advisory Board did not agree that the risk was large, and certainly not large enough to demand emergency power. Whatever the laws at his disposal, the same scientists would be likely to recommend against their use.

Without taking a position on whether or not ALAR should have been banned, I note that the risk of drinking and using water from the treated surface water in most of our big cities calculated using the same pessimistic, conservative procedure, is five times greater because of chloroform than that from ALAR. It is likely to be dominated by the use of water for bathing children, and absorption of chloroform through the skin.

Asbestos provides another example. It is well known that there were large exposures in the workplace 30 and more years ago, that are still producing lung cancer, particularly among smokers. In the environment, exposures are typically over one thousand times smaller. However, 15 years ago some risk assessors began to suggest that there may not be a threshold below which there is no risk and therefore that the lower environmental exposures pose some risk. Moreover they noted that the rare cancer mesothelioma was often caused by asbestos exposure. It has a long latent period. This means that occupational exposures often are too late to allow expression of the disease before death, and children's exposures might be more important than deductions based upon the occupational studies had suggested up to that time.

This realization, urged in particular by Dr. (now Professor) Julian Peto of London University, quickly led to suggestions that damaged asbestos in schools might be dangerous and led to demands of strong action. While I personally think that in most cases it is best to leave the asbestos in place, I note that there is plenty of power for local, state or federal authorities to order removal of asbestos when they want to. What is needed is better use of scientific information so that the agencies can act in the best way for public health and decide whether the enormous amounts they are called upon to spend are indeed likely to be accompanied by improvements in health.

After the federal government has set a reasonable level of safety, I see no reason why citizens should not decide to go further on their own, at their own expense. For example, the EPA call upon schools to study the effect of asbestos in schools -- not necessarily to remove it. It is and should be up to the local group to decide whether to make the expenditures necessary and balance them against the alternative expenditures the school committee can make for the children's welfare. The federal government should not bias the decision by providing money, but must help with good information. I note here that it is

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a common practice to make a bias in favor of removal by allowing a removal contractor to make the recommendation. This is done all too often nowadays.

I would suggest that anyone proposing new laws and regulations show that the proposal will, in fact, improve children's health, or at least have a good chance of doing so. When a criterion demanding that any new regulation be shown to have a significant effect on safety was adopted by the Nuclear Regulatory Commission a few years ago, the number of new regulations dropped manyfold. But all indicators of nuclear safety (such as reduction in the number of small incidents) have continued to improve.

Our agencies are by their nature, adverserial. We ask, and expect, that EPA constantly urge the cause of environmental protection. FDA must push for safe food and drugs. On the other hand, we ask and expect that Commerce and DOE work for cheaper and more plentiful goods and energy. Therefore any balancing of these needs must ultimately be external to these agencies. For financial matters it is the Office of Management and Budget (OMB); we need an office which gives similar scientific advice, balancing and coordinating the scientific matters. Maybe the Office of Science and Technology Policy in the White House could take a lead in this. Congress may wish to ask this -- and provide the budget for it

With all the toxins facing us, it is important that there be research on which are most likely to be especially risky to children. Exposure to children is especially important for carcinogens with a long latent period. A carcinogen with a short latent period, often a "promoter," is usually most important at a late age. Unfortunately there is at the moment little scientific consensus about which of the known toxins are in which category. The "upper limit" of risk must be kept high to encompass these uncertainties, and if we insist on regulating at this upper limit, and to a low risk number, enormous sums can be spent with the likely improvement on health being zero.

Thank you for your attention

Chairman **Miller**. Thank you. Thanks to all of you very much for your testimony and for your help this morning.

Let me just begin by making a statement and see if I can get some reaction. As I stated earlier, this is the second hearing, and I think it took a little longer in this hearing than in the one last week to break down this debate between children dropping dead in the school yard and the apple police.

I don't know whether either camp is right, wrong, or otherwise, in terms of their fears or concerns, but I am much more concerned by what I hear this morning from Mrs. Greenspan, about her child who is walking around apparently in a rather vulnerable state to intrusions by agents that may cause a reaction that is toxic to her in her condition; from Dr. Needleman, and I think from Dr. Schaefer. In your discussion you are also talking about millions of young children who are—I don't know the term that I want to use—but you talked about low levels.

It reminds me of a guy with a hangover. There's nobody I'd rather negotiate with because he's not quite hitting on all cylinders here, and he's going to make a mistake after he has been intoxicated.

I am just thinking here, in terms of sending children out into school, or maybe to the workplace, or whatever their daily activity is, and yet what you are telling me is that because of neurotoxins—Dr. Needleman, you have mentioned lead, Mrs. Greenspan in the case of your daughter, one or two particular things threw her off balance in some fashion or another, children are not going to be sitting as attentively as they might be because of these neurotoxins in the classroom, or what have you.

I have also watched with considerable interest groups that work with real serious delinquents, children who would set their teacher or their parents on fire and have, in some cases, burned down their schools, or assaulted people. I have watched the process of treating these children with removal of neurotoxins from their diets, from their atmosphere, and seen behavior changes.

What do we know about all of this at this lower level? Forget whether every child who eats an apple drops dead, or whether or not we are going to have any pesticides on the market. What do we know about something that may be far more widespread? That is what the three of you have touched on, and that is a lower level of functioning by our children because of the invasion of various possible or real neurotoxins to them.

Dr. **Needleman**. We know a lot about lead, and I think lead may foreshadow things that we are going to find out about other toxins. We have known that lead has been neurotoxic for 2,000 years, and we have known that children have been affected for 100 years. We know that current science has driven the effect level down to this range in which many, many children have it. There may not be a threshold.

A no-effect level has yet to be found, and there is an enormous amount of very good molecular biology and biochemistry, neurochemistry, which shows that the brain has certain targets for lead, for instance, brain protein, kinase C, for which lead is much more an avid seeker than calcium.

It is reasonable then to suspect that the more sensitive methods we use, the more we are going to find effects at lower and lower doses. The way these express themselves in human beings are in disturbed attention, the ability to focus, to execute, to code, and then to shift, when it is appropriate, to a new target.

That is why children display this kind of behavior where they can't sit still or they are very vulnerable to distractions, or, if they finally do focus down on something, they get very sticky and can't shift gears. Deficits are also found in language, and, finally, in what we call intelligence; that is, the ability to do an intelligence test. And the evidence is that it is very pervasive.

Chairman **MILLER**. Dr. Schaefer.

Dr. **SCHAEFER**. I think Dr. Needleman made a very good point when he indicated that we have known about lead being toxic for 2,000 years. We have 65,000 chemicals in commerce, and we really understand very little about the toxicology of these substances.

So when you talk about concern about our children being exposed to low levels of substances that may be having some type of very subtle effect on their behavior, when you think about lead as an example—and we have certainly done a lot about it in recent years, and much of that is due to Dr. Needleman personally—I think it is clear that we really need to do more research. We need to do more testing to determine whether there are other substances like lead out there.

It is totally conjecture, but if you have that many substances in commerce, you have to assume that there are some other bad actors out there that we don't know about. The question is whether Congress and the public think it is worthwhile to try to identify these substances.

Chairman **MILLER**. Let me ask you, from a layperson's point of view, and as Mr. Sikorski said, we are here as parents also. When you say that three to four million children have toxic levels of lead, what are you telling me?

Dr. **NEEDLEMAN**. That is the official Agency for Toxic Substances—

Chairman **MILLER**. I understand. But when you say "toxic," you are saying—

Dr. **NEEDLEMAN**. Over 15.

Chairman **MILLER**. No, no, no. What is the impact?

Dr. **NEEDLEMAN**. I think a major portion of those children have disturbed attention, that if you scaled them by teachers' rating scales, the teachers would report that they don't do as well as other kids. The work that we have just completed shows, I think very strongly, that 10 or 11 years from now they are going to be worse, that they won't do as well at school, and they probably won't be able to make as good a living.

Chairman **MILLER**. One of the things we try to do in the select committee is look today plus 10 years, or today plus 15 years, because we spend a great deal of time working with children, and, of course, you want to know where these kids are going to end up, their ability to contribute, and will they contribute, to our society.

I am trying to think of what else it is that we have in the childhood population that would detrimentally affect three or four million children.

Dr. **Needleman**. That is why I said I think it is the most serious problem in American pediatric medicine.

Chairman **Miller**. Does anything come quickly to mind?

Dr. **Needleman**. Malnutrition.

Chairman **Miller**. Malnutrition is the only thing I could think of that would affect——

Dr. **Needleman**. Injuries, perhaps.

Chairman **Miller**. Yes, I guess childhood accidents. What you are suggesting—and somewhere you mentioned 400,000 newborns.

Dr. **Needleman**. Newborns. Per year.

Chairman **Miller**. I mean, we are launching candidates for failure.

Dr. **Needleman**. Exactly.

Mr. **Wilson**. Mr. Chairman, I wonder if I could—

Chairman **Miller**. Just one second.

We try to think that sometimes this committee and our concerns and our work is about increasing the opportunity for success among our children, whatever endeavor they choose and what we hope for them, and everything else. But here, I mean, you are just sending out into society children with a toxic level which you are telling me, as a layperson, is some diminished capacity, especially with relationship to their learning experience in school.

You are right; you said "staggering" in your testimony. That is a staggering, staggering figure in terms of, as we look around, how—we keep asking for more productivity, more productivity out of each and every one of us to keep the American standard of living alive and well. It starts to get a little complicated down the road if we are launching these children at these levels.

Obviously, we are taking some actions to reduce those numbers, but there is also some indication that a lot of the actions we have taken have not directly related to the decrease in children.

Dr. **Needleman**. That's right. There was an article in the New York Times somewhere in the past month or so about industrialists who are very concerned that they cannot staff a modern factory assembly line because it is much more demanding work. You have to know how to program a computer, execute complex steps in assembly, and they are finding that American workers are not matching up to foreign workers.

Chairman **Miller**. We have heard this in the committee. We have heard it from the business councils and various people coming to us and saying that when they look down the road, they are not quite sure how they assemble the work force necessary for America in the next century. But I guess what I am saying here is, you are giving us a little bit of a look behind those figures and saying that this is going on almost unconsciously with respect to millions of young children as they are trying to gain those skills.

Dr. **Needleman**. I think it is quite relevant to that specific problem.

Chairman **Miller**. Mr. Wilson.

Mr. **Wilson**. I don't want to disagree with anything Dr. Needleman said, but I want to emphasize one or two things. One is the importance of comparing things carefully. Recently there was a meeting on cadmium, which is a carcinogen probably, and in Sweden a leading person from the Sweden Department of Health said, "We have abolished the use of cadmium in Sweden." And we said, "Well, what did you do? You had cadmium for use; you must have done something instead." They said, "Oh, yes, we replaced it with lead."

Of course, that was exactly the wrong thing for him to do for Sweden. So, without thinking carefully of alternatives, you can easily do the wrong thing, as they did in Sweden.

Secondly, I do want to mention that the lead levels have been coming down. At the time I was born in England, I think everybody had the health defects that Needleman was talking about, not just 15 percent.

Dr. **Needleman**. Look what happened there.

Mr. **Wilson**. There is a recent book saying lead poisoning destroyed the Roman Empire, and you might argue the lead levels in England destroyed the British Empire. The point is, it was true everywhere.

One thing we want to emphasize is not the past, that we have in fact survived the past, we have survived a society which had fairly high lead levels, but we want to do better, and we want to do better not just with lead but the other things we don't know about. It is very important to do better than we did before.

Chairman **Miller**. We have survived it. My concern is that when people go to invest money or they choose investments they decide, what is the cost of lost opportunity in another investment. I am concerned that when we launch 400,000 children, in the case of lead poisoning, or we have children that have the problems that are confronting society, of Mrs. Greenspan's child, there is a lost opportunity cost.

As she said, it is not just that there are three million stupid kids, it is that there are some kids that are going to be on the margin between superior performance, which may provide a great return to society generally, and they won't get that opportunity, and they haven't done anything. They just showed up in the wrong neighborhood. That's a lost opportunity cost that concerns me.

It is not that, "Well, Jesus, you know, other societies survived for 2,000 years we've known about this." I want to be better than those other societies, and I want my kids to be better than those other societies. So I can't even figure out the opportunity cost.

Mr. **Wilson**. I completely agree with that.

Chairman **Miller**. We talk about people who come along once in a generation or once in a lifetime, and now we are snuffing out the opportunity for some of those people to come along.

Mr. **Wilson**. There is one other point one has to make. We don't really know all the reasons why the lead levels fell. One of them is better nutrition, we are fairly sure, though it was a combination between iron deficiency and lead that people have argued about. Better nutrition in all ways is good. We have to understand exactly what is the best way of getting it all down.

The effect is so big, the number of people exposed is so big, if we just said we are going to go all out to reduce lead exposures in this country to, say, one microgram per deciliter in the world, there just isn't enough money in the country to do it at the present moment and with the present methods we know what to do.

Chairman **Miller**. Mr. Sikorski.

Mr. **Sikorski**. Thank you, Mr. Chairman.

Mr. Wilson, I agree with much of what you brought with you today. I don't like OMB making decisions that are scientific or even moral in basis. There are very few people over there—most of them are economists and lawyers. George Bernard Shaw said, "If you take all the economists in the world and lay them end to end, they would never reach a conclusion." I am a lawyer. He said, "If you took all the lawyers in the world and laid them end to end, it would be a good thing."

That's who you have making these scientific decisions, and I agree with you. I disagree with some of what you said. I don't think the only role of the Federal Government in the area is to subsidize professors in their research and end it there. And I have questions about this $100 billion that industry is spending. If you mean less produce because they are using fewer chemicals, I can see that.

I did want to focus on—you used the ALAR example. I know you didn't do this purposely, but you molded that situation to make a point here. What you say is not accurate. You said, Mr. Moore, who I disagree with but will defend in this case, could have done—you disagreed with Jack Moore when he stated on "60 Minutes" that the laws did not permit him to act fast. I disagreed with him when he said the laws did not permit him to act. I thought he could act.

You went on to say he could have asked for emergency cancellation of registration, "jaw-boned" the apple industry, advised state commissioners of health to use their emergency powers, then you go on to say, "I believe that his only hesitation in using emergency powers was the knowledge that many scientists on EPA's Science Advisory Board did not," underlined, "did not agree that the risk was large, and certainly not large enough to demand emergency power. Whatever the laws at his disposal, the same scientists would be likely to recommend against their use."

That ain't true. Mr. Moore and the Scientific Advisory Panel agreed that the risk was large. The Scientific Advisory Panel said, and I quote, "After weighing the risks and benefits from daminozide use, the agency has determined that continuing the current registration for food uses of daminozide present unreasonable risks. Therefore, the agency is proposing to cancel all food uses of daminozide." That was not a disagreement.

Mr. **Wilson**. I think that wasn't unanimous.

Mr. **Sikorski**. Well, the Science Advisory Panel did agree that the risk was large, and certainly was large enough to demand emergency power. That's not what you said.

Moore did send letters—the "jaw-boning" aspect. You recommend he "jaw-bone". He did send letters to the apple and peanut growers warning them of the dangers and requesting them to voluntarily cease use, to no avail. The state health commissioners did act; in the abdication of Federal activity, they did act.

The reason Moore couldn't or didn't act, or didn't want to act, was that he had to prove what is called imminent hazard under the laws, which hasn't been defined in pesticide case law, yet he feared tremendous litigation. I said, that's his job, and I think we are in agreement there.

Mr. **Wilson**. Right.

Mr. **Sikorski**. ALAR is an acute hazard with harmful effects evident only in the long term. It was the immediacy of the risk that was at issue, not the risk.

I want to give you a chance to respond.

Mr. **Wilson**. I wonder if I can modify my comment just to address those points.

Mr. **Sikorski**. Sure.

Mr. **Wilson**. My implication that there were some scientists on the advisory committee who disagreed with that, that is actually a fact, because I have talked to them.

Mr. **Sikorski**. There were a couple.

Mr. **Wilson**. The question was, the way that would enter is if Mr. Moore had gone to public hearing—if the thing had gone to court, those scientists would probably have wanted to testify against him. The existence of people who are willing to testify on the other side was very important. If there was unanimous agreement, then it would not have been opposed in court. No scientist would have opposed it in court. That was the point I was trying to make.

The agreement of the EPA—the size of the risk was approximately the size I mentioned in the following paragraph.

Mr. **Sikorski**. I get further into this in the sense that if you are proposing that only when science is unanimous—you know, Kierkegaard, Tolstoy, even Frank Sinatra, in some of his songs, never came up with absolute knowledge. There ain't such a thing, theologically, in existence, that there is an absolute, definite human conclusion or scientific fact.

You will find a scientist, like an economist or a lawyer, who will testify in court for your purposes. I can get them.

Mr. **Wilson**. I am far from proposing that, Congressman. In fact, my point was, I disagreed with Mr. Jack Moore. If he had the evidence he had with him and wanted to do that, he should have just gone ahead and done it and had the whole thing out in the open.

Mr. **Sikorski**. Yes. There we are in agreement, and I think you hit the point that needs to be stressed. If the Government of the United States does not deal with interstate commerce products, if the Environmental Protection Agency does not protect the environment, if the Food and Drug Administration does not protect the food supply, and if the industries do not protect their commercial rear ends and their legal rear ends by being careful about what they market, guess who regulates in America? Grass roots consumers.

And they do it crudely and cruelly, and you get apples pulled off the market when applesauce and apple juice should be pulled off the market. And you get apple growers who don't use daminozide, never used daminozide, bankrupt when those that have continue happily on their way. That's what happens in those—Dr. Needleman on the lead thing.

Mr. **Wilson**. I completely agree with you, Congressman.

Mr. **Sikorski**. Thank you.

Mr. **Wilson**. I want to say I am one of the scientists who, in 1978, publicly and in scientific papers said I believe daminozide is a carcinogen according to all the rules of EPA.

Mr. **Sikorski**. Thank you.

Dr. Needleman, most of us, if we were given a choice of injury to our body or injury to our mind, we would choose injury to our body. Terrible choice, but I think most of us would do that. And most of us, given the choice, injury to our children or injury to ourselves, we would take it upon ourselves.

That is why lead is so terrible; it affects the mind. It is terrible on the body; it stunts. It doesn't get out. It frees itself up and hits again someplace else, physiologically. But intellectually it does terrible damage. It is tough on adults, especially vulnerable adults, pregnant women and others, but it really hits our kids.

I want to commend you for your efforts in this area, your efforts to take your scientific background and use it to assist policymakers in doing something good. The numbers don't need reciting, but 400,000 new babies each year hit by this, we are losing two million super-Americans. We are losing two million geniuses or near-geniuses from international competition, from national security, from cleaning up the environment, from staffing our major universities and colleges, and the rest of it, and that is a scandal.

Thank you for your efforts.

Dr. **Needleman**. Thank you for your comments.

Mr. **Sikorski**. Thank you Mrs. Greenspan.

Mrs. **Greenspan**. In response to your comment, Mr. Miller, about the effect of low levels of air pollutants on children, I just want you to consider one quick scenario. Let us take Dr. Needleman's three million children with high lead levels and put them in school in the wintertime where there is very little air circulation because our energy consciousness has made us decrease the air flow in the buildings.

Let us then say this school's number has come up, and they are going to paint indoors for the next month. During that month, the pesticide man comes because it is his time to fumigate the building for roaches. Each night maintenance men are cleaning, using mopoline on the floor. The mopoline has in a kerosene type base, and those fumes are dispersed throughout the building, together with those from the xerox machine, and the glues, etc.

When you finish, I think you not only have a low level, but a high level of many toxins. And I don't think there is any scientist in this room that can prove that those toxins don't have an effect on the children. I can not prove that they do, because there is no research, but consider that these children are already impaired because they have high lead levels.

I would strongly urge everyone here to at least take a look. When you take a look and you see that there are some problems, because I am sure you will, then set standards that really help children.

When you have school systems putting on asphalt roofs, creating fumes in buildings, and causing children to have headaches, the children cannot learn and they are not well.

Chairman **Miller**. Mr. Walsh.

Mr. **Walsh**. I thank the chairman.

I must say I enjoyed the gentleman's reference to Kierkegaard, Tolstoy, and Sinatra. I guess the common thread there is that they all did it their way. Scientific achievement certainly wasn't the common thread.

I do agree that it would be foolish and impossible to wait for unanimity in science before we decided to do anything in this world. The question then, when you do not have unanimous agreement on a chemical or a risk, is, how do you assign acceptable risk? I would like to just ask Mr. Wilson a little bit. You mentioned the Delaney clause and that that was an unacceptable level of perfection, I guess, if you will. How do we get at assessing risk? How do we get at this idea?

We deal with it in Agriculture, and I know they deal with it in the Energy and Commerce Committee. How do you get at assessing risk and what is acceptable risk? How do you determine what chemical has what acceptable level of risk, and how do you assess that versus the loss of nutritional value? If everybody stopped eating apples, what do we lose? If everybody continued to eat apples with a certain percentage of a certain chemical on it, how do you get at that?

How do you quantify it? How do we legislate it?

Mr. WILSON. It is very hard to quantify a comparison there, but I think there is no doubt that when the question came, should we do anything about ALAR, even those people who felt the risk is not terribly large—as I say, if you calculate it in a similar way, it is less than that of drinking water in Washington, D.C., even for children—the question would then come up: Do you ban it?

The question immediately comes up: What is the use of ALAR, and is there any disadvantage in banning ALAR? Most of the people who were thinking about that question felt there was not a very strong point in having it there, so that particular management decision came up.

The question is, how does that decision get made? My contention is you have to have the information available to the people to make that decision. The man making that decision not only has to know roughly what scientists think about that, the magnitude of the risk, and he also has to have in front of him what the risks are. Then you do the balancing. Sometimes it is the federal government that does the balancing.

Ninety-five percent of the risks taken in our society are taken by individuals in the marketplace and other places. The more information they have available to them on making those judgments the better. They need to know, for example, should you go into a building which has bad ventilation? We have just heard the problems of the indoors. They are all real. If you have bad ventilation, they are exacerbated. Those are decisions we individually make. The important thing is that information.

Now, how to get scientists' agreement? There is a beginning technique of trying to get understanding and quantification of expert judgment. One of the problems is that most scientists sit in an ivory tower and don't think, like Dr. Needleman does or I try to do, of what is the effect of my science on general public policy issues. Then you ask them, and you won't get a useful answer.

You have to train them to think in this particular method. That is a process now known as solicitation of expert opinion. You have them sitting down for three or four days with you so they understand that particular problem, then they state their opinion so it addresses the issues you are trying to address. Professor Keeney, at the University of Southern California, is probably the country's expert in that particular matter.

Mr. **Walsh**. Thank you, Doctor.

I would just like thank Dr. Needleman for his comments and his work in the area of lead poisoning. I don't think there is any greater risk to America's children, especially in urban areas, than the risk of lead poisoning, and I certainly support your efforts, congratulate you for your hard work and for raising America's consciousness to this problem.

I would like to ask just one question, and that is, what effect have you seen in your studies of the effect of the legislation back in the early 1970s to eliminate lead in paint and in gasoline and other substances?

Dr. **Needleman**. The Lead Paint Act was a good act, but HUD just dropped it. I mean, the number of houses that have been abated is vanishingly small. That article in the New York Times by the Chicago Bureau Chief deals with that quite authoritatively, and I can tell you that is accurate.

The struggle to remove lead in gasoline was a very intense one, in which the medical, public interest, and public health community fought very hard and achieved it, and lead levels in American children and adults have come down in very tight correlation. It may be in the OTA document, there are some plots of blood leads versus gasoline lead sales in different communities, and air lead levels, and the correlation is like .9.

So that is a very encouraging event that we can take a step and see in a matter of a couple of years a beneficial outcome.

Mr. **Walsh**. So the real problem that remains is removing or replacing, or in some way abating, the lead paint that still exists in those two million homes?

Dr. **Needleman**. There are children living in homes that if instead of lead it was cholera or some other toxin, we wouldn't allow it. Every Wednesday I have a clinic in Pittsburgh, and I see those kids. I tell the mothers, you know, we are going to try to find you another home. There is precious little money to relocate these people, so I talk to them about washing their hands and not letting them suck their thumbs. Well, tell a mother not to let her kid suck his thumb.

We do what little we can, but there is a reality that a large segment of American children are living in unsafe circumstances, that can be taken care of. Secretary Mason is seriously regarding this. It is going to cost billions of dollars, but the payoffs will be enormous.

Mr. **Walsh**. What a horror that must be for a mother to know that her children are growing up in an environment that is affecting their ability to think, and their ability to function, and their ability to learn, and not be able to do anything about it.

Dr. **Needleman**. Exactly.

Mr. **Walsh**. Thank you, Doctor.

Dr. **Needleman**. Thank you.

Chairman **Miller**. Mr. Lehman.

Mr. **Lehman**. Thank you, Mr. Chairman.

There was a mention of organic food and also of tap water. How many of the four panelists eat only organic food in their homes?

How many of the panelists only drink bottled water in their homes? Two of you.

If I went out and bought organic food, randomly, from 10 different organic food stores, what are the chances that I would be buying genuine organic food rather than something that the store just claims is organic? Does anybody have any idea what kind of control the government has over the validity of so-called pure organic food?

Mrs. Greenspan. There is someone else here, who can probably answer that for you later on, Jay Feldman. My understanding is there isn't any regulation. There are some states, the State of Maryland, has an organic food——

Mr. Lehman. But anytime you write "organic" on a carrot, no matter where it comes from, and put it on a shelf in a health food store, it is automatically organic food. Nobody knows whether it really is or not. The FDA inspects the meat-packing plants, but they don't inspect the so-called organic food.

Mrs. Greenspan. My understanding is they don't do a very good job with the meat plants either.

Mr. Lehman. It is an honor system as much as anything else; right? How about bottled water? Does anybody know where bottled water comes from really. One of you only drank bottled water in your home besides Mrs. Greenspan. Is it really bottled water?

Dr. Needleman. I have a filter that is reported to remove 90 percent of the lead and chloroform. Have I tested it? No, I haven't. But it is a small investment.

Mr. Wilson. I think Dr. Needleman does better than drinking bottled water, because the important route of exposure is not drinking water with chloroform and other organic materials in it, but dermal absorption. Organic materials go through the skin very readily when you bathe, and they come out into the air when you take a shower, and you breathe them in. That is the biggest route of exposure.

So Dr. Needleman has done the right thing; he is filtering it and taking them out rather than merely switching to bottled water.

Mr. Lehman. Should I buy bottled water and drink it in my home only, or should I just go ahead and use tap water? Can you answer me that?

Dr. Needleman. I think it is a matter of personal choice, and I have made mine.

Mrs. Greenspan. I also have a whole house filter to take the chlorine out of the water for me.

Mr. Lehman. The main food of children is milk. Nobody mentioned whether the cows should be fed organic food or not. Is that a problem with the cows eating bad food and producing bad milk?

Dr. Needleman. I have not studied that.

Mr. Wilson. I don't think there is any doubt that many of the pesticides fall on the materials that are eaten by a cow and they then come out in the cow's milk. We are very fortunate in the metabolism of a cow. The worst of the poisons we have is actually a natural one, aflatoxin B-1. It is present on nut products; fortunately, cows convert it into a different isomer of aflatoxin, which is much less dangerous.

Nonetheless, a lot of pesticides do come through the milk.

Mr. **Lehman**. I visited two landfills—talking about lead poisoning—I visited two landfills recently in my area, and a major portion of the landfill is discarded acid lead batteries leaking into the adjacent water. What should we do about that?

Dr. **Needleman**. It should be disposed of in a way that is safe.

Mr. **Lehman**. Where?

Dr. **Needleman**. I think the best thing to do is put it back deep in the earth where it started out, away from the aquifer.

Mr. **Lehman**. Should we have a federal regulation to bury the acid lead batteries someplace?

Mrs. **Greenspan**. Batteries should be considered hazardous waste. We already have regulations to deal with hazardous waste, and it should be dealt with through that mechanism.

Mr. **Lehman**. It is still not being very well controlled. Right now they are trying to dredge the Miami River, which hasn't been dredged for—some battery manufacturers are on the river, and nobody wants the sludge. Where do you want us to send the sludge to?

Mrs. **Greenspan**. You are pointing out, and I agree with you completely, we have a tremendous problem in this country with environmental pollutants.

Mr. **Lehman**. I have known a lot of people in the battery business, as manufacturers of rebuilt batteries. Has there been any study on the effects of breathing lead residue on people that work in lead acid battery companies and on the health of their children?

Dr. **Needleman**. Yes.

Mr. **Lehman**. For the record, could you put something in as to what happens to people that work in battery companies?

Dr. **Needleman**. Sure. It was by Edward Baker, who was formerly assistant director of NIOSH, Baker, Philip Landrigan, and others, on the effects of exposure in smelters, secondary smelters, and the effects on their children, because they bring the lead home on their clothes and on their hair.

Mr. **Lehman**. And was the Roman Empire destroyed by storing wine in lead vessels?

Dr. **Needleman**. That is a thesis.

Mr. **Lehman**. How important do you think that was?

Dr. **Needleman**. I don't do that kind of science.

Mr. **Lehman**. A couple other quick questions. You said malnutrition was one of the great problems of health in children, how about obesity?

Dr. **Needleman**. It is a serious problem, but not of the same magnitude.

Mr. **Lehman**. Not in the same category?

Dr. **Needleman**. Right.

Mr. **Lehman**. We heard a lot about the effect on children of living in and around high voltage transmission wires. That wasn't mentioned, and that is a threat, if it is valid, to the health of children. Do you have any opinions on what happens to children that live in the vicinity of high voltage transmission wires?

Dr. **Needleman**. Well, there are a couple of epidemiologic studies. It is a very difficult problem to study. There is reason to be concerned, but that is about as far as I would want to go.

Mr. **Lehman**. Let me know if you find a place to put some of this toxic waste, but "not in my backyard". I think that is one of the biggest problems we have right now.

Mrs. **Greenspan**. Mr. Lehman, perhaps if we could get some of the lead levels out and some of the IQs could be raised, these very smart people, if they become aware of the pollutant problem, can then solve the problem by making things less toxic.

Mr. **Lehman**. Maybe if some of them got the lead out of their bottoms, it would be better, too.

Mrs. **Greenspan**. Agreed.

Mr. **Lehman**. And get to work on this problem. Thank you very much.

Chairman **Miller**. Mr. Durbin.

Mr. **Durbin**. Thank you, Mr. Chairman.

I apologize for being late, but I appreciate your attendance here. I just have a few questions.

Will the horrors of the environmental problems of Eastern Europe be of any assistance to us in assessing the long-term impact of chemicals on children, and adults, for that matter?

Dr. **Needleman**. I have been asked to look at some of those things, and may get involved in Eastern Europe. These are extraordinarily high doses. I think the big lesson to be gotten from that is that all occurred in 45 years. Since the end of World War II, these outrages and public health disasters have been created. I think the most important instruction to be gotten from it is, we had better be very careful ourselves, because, if we do not attend to things, we could end up in similar circumstances for certain pollutants.

Mr. **Durbin**. I might add that in my home area, it is an agricultural area, and the farmers are as concerned as anyone else about the chemicals which they use for their livelihood. We are very proud of the productivity, when it comes to corn, soybeans, and wheat, and, of course, it is a very expensive undertaking for a farmer to add chemicals to stop pests or weeds, or whatever it might be.

There is a feeling in my part of the world of confusion and frustration about this whole subject. We just don't know what is dangerous and what isn't. We don't know whether parts per billion is enough to worry about or parts per trillion. We don't know whether three cases of neuroblastoma, a rare cancer, showing up in a town of 10,000 people that I represent has been caused by an old coal gasification plant—and there are hundreds of them across the United States, abandoned coal gasification plants from the old days of the gas lamps, with coal tar residues, unfortunately many of them in very perilous positions.

How do we address this general frustration where finally people throw up their hands and they say, "I can't make a move. I can't live under a high-power line. I can't feed my kids the food that comes out of the grocery store. I can't give them what they want to eat. I can't sit them down in front of the television. Some scientist is warning me it's killing them."

I think they finally reach a point, most people do, where they say, "I'm going to start ignoring it," except for the most egregious situations: the exposure to lead paint, for example, there is no argument there. There are certain things that are just across the line. But if you start being sensitive to the possibilities, I don't know if you can lead a very normal life in America today.

There is not a day that goes by that there is not another warning coming down about something else. How does science address this? What is your responsibility?

Dr. **Needleman.** I think we have a responsibility, as scientists, to make good measurements and then to tell the truth. When pollutants reach that scale or dispersion that people have limited utility to help themselves because of their limited options about where to live or because it is pervasive in the air, then it is a public health problem that demands a public health solution. That is up to Congress, and it is up to DHHS to act.

My own calculus puts heavy weight, in circumstances of uncertainty, on having wide margins of safety, because the history of science is that the more we learn, the more we find out that many things are dangerous. It was only in my lifetime that a blood lead of 60 was considered okay. If a kid had a blood lead of 59, you followed them; you didn't treat them. Now we are talking about blood leads of 10 or below. That is not in a very long time in the history of the world.

I think the same kinds of experiences will be recapitulated with other toxins, not every one of them, but for many of them. Better science is going to find effects at lower and lower doses. It is unusual to find that good science says, "we found that we overestimated the risk". So my own particular bias, and I think it is an educated bias, is that we should be building in large margins of safety until the science sharpens the focus on the question.

Mr. **Durbin.** As you build in the wider margins and you start bringing in more factors to be considered, you can appreciate the level of frustration of conscientious people, trying to draw a line as to how you live a normal life under those circumstances.

Dr. **Needleman.** Yes. One shouldn't get panicked about it. I mean, I am not terrified; I am concerned about many of these things: pesticides, mutagens, teratogens, behavioral teratogens. I think these are real risks that have to be addressed, and one works at them. I think one should recognize that if you put out a biologically active substance, if it affects insects, it has a high probability of affecting similar chemical receptors in humans.

Now, there are repair mechanisms in living organisms, but they don't always work. That is the current state of our risk. Everything that is biologically active on a fungus or an insect is a candidate to be biologically active in a human being.

Mr. **Wilson.** I would like to just emphasize something Dr. Needleman has said. You had an implication in your question that people are finding it more difficult to live a more normal life than they used to, perhaps. I want to insist that in fact the reason they have raised lots of questions is, we are trying to do more than we ever did when I was a boy and that there are many more scientists, looking in much more detail, like Dr. Needleman.

As he said, at the time I was a boy, 100 micrograms per deciliter of lead, there was a question was that dangerous or not. We now do not even consider whether it is dangerous or not; we do not have it. So we are doing better, and more and more questions automatically come up because we are doing better; it is not because we are doing worse. We must recognize always, don't panic because we are asking more questions, because we are a lot better off than we were before.

Mr. **Durbin**. Could I ask, and perhaps this is beyond your area of expertise or even beyond this hearing, but does the subject of environmental tobacco smoke or secondhand smoke come into this discussion?

Dr. **Needleman**. Absolutely. One of the best examples of a serious threat, it is one of the most serious threats, and it is also an example of how fast we have come to understand that it is. Science has demonstrated that children in homes where parents smoke have higher respiratory disease and other bad outcomes.

Mr. **Durbin**. I have a personal interest in the subject. In some of the legislation that I have worked on, we find it increasingly difficult—and most of the reasons are political—to develop a sound policy at the Federal level to deal with environmental tobacco smoke. We have made some progress.

It just strikes me as curious, and maybe this is a rhetorical question, that we would brand some parents as abusive parents for treatment of the children and yet look beyond the fact that some of the habits of parents, such as chain smoking in the presence of small children or during a pregnancy, can be just as abusive if not more abusive than some of the things that are being prosecuted today.

It may be impossible to police, but it suggests to me that, in terms of a national public health campaign, we have to be a lot more aggressive than we have been in letting people know how dangerous it is to have secondhand tobacco smoke, particularly in the presence of small children.

Mr. **Wilson**. I would like to say, I am very much pleased, to notice a change. The first time I testified on risks in this House, about 12 years ago, the chairman was smoking, and several other people in the room were smoking. I am glad I see that no one is smoking in this room. I think this is a major improvement in public health, for which we can all be thankful.

Mr. **Durbin**. I hope that some of you flew here, and if you did and there was no smoking on the planes, Congress is responsible for that, too. So we are coming a long way.

Chairman **Miller**. Mr. Durbin did that, too. Mr. Durbin has never met a cigarette he liked.

Dr. Schaefer, you made a series of recommendations for the Congress, in terms of things you said we could do— you were very gentle, thank you. Let me ask you, what is next for you? Is there a further distillation of this first report to provide us some additional blueprints? Does the office have under active consideration pursuing this?

Dr. **Schaefer**. In terms of neurotoxicity, the project is essentially complete other than follow-up activities. The Senate Committee on Environment and Public Works requested a series of studies on noncancer health risks, and OTA presently has a study underway on related to immunotoxicity—effects on the immune system. After that, I understand they are going to move on to the pulmonary system, and then, in the subsequent report, they will examine the effects on all the other organ systems collectively.

So they will cover the noncancer area pretty well in the next couple years or so.

Chairman **Miller**. If I can cross over to Mrs. Greenspan's problem with her child, in the sense that the child has childhood diabetes; right, if I am correct?

Mrs. **Greenspan**. Yes.

Chairman **Miller**. Because of your very close monitoring, you think you have made relationships between various activities and materials and the toxins. Dr. Schaefer, what do we know about that? What do we know about a number of children that come into this world with a series of problems and the interplay of other agents in our general environment in making their lives more difficult? Have we even begun to look at that?

Dr. **Schaefer**. Yes, we have. It is an appropriate and timely question. Yesterday, OTA held a workshop related to their immunotoxicity report, and one thing they talked about was this question of multiple chemical sensitivities. It is a controversial issue, as you may know.

Chairman **Miller**. Yes.

Dr. **Schaefer**. It is unclear how that is going to come out. There is considerable debate right now about the physiological and biochemical basis of sensitivity to chemicals, but it does seem to appear that there are people in the population, for whatever reason, who have an increased sensitivity to toxic substances. Unfortunately, her daughter may be one of them.

Chairman **Miller**. To the degree of serious disabilities, in some instances; right?

Dr. **Schaefer**. That is the argument. It is an active area of research, but it deserves much more attention. In fact, with respect to chemical sensitivities, there is a special problem because the medical establishment is somewhat reluctant to get into this area.

Chairman **Miller**. Somewhat?

Dr. **Schaefer**. You know about that.

Put yourself in the place of a researcher who would like to look into it, who are you going to go to for funding to support your research project? NIH may not look upon it too positively.

Chairman **Miller**. Try just being a practicing physician and suggest that this may be a problem.

Dr. **Schaefer**. Yes. This is an area that deserves considerable research.

Chairman **Miller**. That is very encouraging. I am delighted that OTA is doing this. I assume that the Commerce Committee, at some point, like the Senate counterpart on the environment, will be looking at the results of this. We don't legislate in this committee, but, clearly, you have made a series of recommendations that should be taken under active consideration as the various committees of jurisdiction—you mentioned some actions with respect to FIFRA and others—as we look at the reauthorization of those acts.

Thank you very much, all of you, for your testimony. It has been very helpful and also very interesting.

Our next panel will be made up of Dr. Susan Pollack, who is an instructor in community medicine and pediatrics, Mount Sinai School of Medicine, and Dr. Chris Wilkinson, who is the managing toxicologist for RiskFocus, Versar, from Springfield, Virginia, and

Jay Feldman, who is the national coordinator, National Coalition Against the Misuse of Pesticides.

Welcome to the committee. Like the previous panel, your testimony and supporting documents will be placed in the record. You can see this is raising a number of questions here, so the extent to which you can summarize would be appreciated. We will take you in the order in which I called you.

Dr. Pollack.

STATEMENT OF SUSAN H. POLLACK, M.D., INSTRUCTOR, COMMUNITY MEDICINE AND PEDIATRICS, MOUNT SINAI SCHOOL OF MEDICINE OF THE CITY UNIVERSITY OF NEW YORK, NY

Dr. POLLACK. Good morning, Chairman Miller and members of the Select Committee.

My name is Susan Pollack, and I am a board-certified pediatrician and an instructor in community medicine from Mount Sinai in New York City. With Dr. Landrigan, we have been working on a two-year study of the health hazards of child labor, prompted in part by the fact that over 1100 children in New York State every year were getting Workers' Compensation for injuries incurred at work. This project has been supported by W.T. Grant Foundation and by the National Institute for Occupational Safety and Health.

We submitted some written testimony, but a fair amount of what I am going to say today is not in that, in hopes of adding something more than what was in that. We are very pleased to have been invited. Thank you.

Basically, as has been discussed, children can incur exposure to potentially hazardous chemicals in a variety of ways, including contamination of drinking water, contamination of food, contamination of soils, and air pollution. I think the point that has been brought up here this morning is, once you put chemicals into those environments, it is sometimes pretty difficult to get rid of it or to get it out.

Children can also be exposed to chemicals at home, on lawns. Parents still bring chemicals home from work to their children, lead included among those, despite the fact that we know so much about lead. Lastly, children themselves may incur exposures when they go to work. There were more than four million children legally employed in 1988.

I say that because a lot of people think children don't work, but it is legal for 11-year-olds to deliver your newspaper and 14-year-olds to work in grocery stores and 16-year-olds to work in restaurants. However, the Fair Labor Standards Act, which was passed in 1938 by Congress, does regulate the hazardous exposures which children are not allowed to incur, that includes both machinery and toxic chemicals.

Despite this, children have a lot of exposures. We have seen children in garment sweat shops in New York City, particularly in leather and belt factories where 14-year-olds spend long days in rooms that are not well ventilated. When you walk in there, it reeks of solvents and glues and your eyes burn, and children are spending whole days in there. We know that solvents can be neurotoxic if you have enough exposure over a long enough duration of time.

We have seen children who have been working for extended family businesses ripping out asbestos in illegal manners, so that even though we know asbestos is a human carcinogen, children sometimes are still incurring that exposure. A lot of children pump gasoline in service stations. In unleaded gasoline there is four to five percent benzene. We know benzene is a human carcinogen; it is a proven human carcinogen.

Certainly, in places like New Jersey where they have mandatory recapture technology for eliminating some of the fumes, children breathe less of that, but their dermal exposure is still an issue. Since we know it causes leukemia in adults, we should at least be concerned about what children are doing around it and at least try to get them to limit their dermal exposure.

Probably, the thing I would like to talk most about this morning is just that children in a number of occupations have significant exposures to pesticides. One of the ways in which that is true is in lawn care and landscaping. It used to be, if you did lawn care as a high school student, you mowed somebody's lawn. But in just the last 10 years or so, I think that lawn care has become more and more a chemical industry, and some of the people working for those companies are sometimes people under 18, who are not supposed to be working with those chemicals.

The other thing is that they don't wear very good protective equipment. People who spray trees wear shorts and short-sleeved shirts and nothing on their heads, and the spray rains down on them. Plus, if you are a person mowing somebody's lawn that was sprayed the day before, you may not even know that you are mowing somebody's lawn that was sprayed by ChemLawn or some other company the day before. Therefore, you may not be able to take appropriate steps to protect yourself, because you may not be aware that it was sprayed.

Another large group of children who have significant exposure to pesticides, and I think illustrate a lot of the problems, are migrant farm worker children. There are child labor laws which prohibit hazardous exposures. Migrant farm worker children fall outside of many of the child labor laws, and that is part of the problem, but it is not the whole problem. They work in the fields for long hours, often far exceeding what is legal, and that increases their duration of exposure time.

Despite the fact, also, that there is a field sanitation standard, many migrant farm workers still don't have basic facilities for going to the bathroom or washing their hands, and they don't have water in the fields, so they can't wash their hands before eating, which, again, causes them to have more exposure because they are eating whatever is on their hands after they have been picking.

As part of our work, we have studied 50 working migrant farm worker children in upstate New York. These are U.S. citizens; these are not illegal children. These are Americans. They are Mexican-Americans who live in Texas half the year and live in New York State the other half of the year. They work in the fields during the day and go to school at night, even during the school year, for the time that they are in New York State.

Of these 50 children, four boys under age 18 have been mixing pesticides, even though that is specifically prohibited under the Fair Labor Standards Act. They were 17, 16, 15, and 13 years old. The 15-year-old is the crew leader's son. Three boys also applied pesticides, and, again, this is prohibited because it is considered a dangerous occupation and children are therefore allowed to work but not supposed to work in dangerous occupations. That also included the 13-year-old.

Pesticide manufacturers specify reentry times after application on the field, and you are supposed to abide by those to prevent people from being exposed. When I was a medical student working on the Eastern Shore of Virginia, we saw people all the time in our clinic who told us that they worked in fields where the tomatoes were still wet. On certain days, they would tell their children not to eat tomatoes because there were wet pesticides still on them.

The children walked barefoot in the fields, so we worried about that, but we didn't have a very good idea how prevalent this problem really was. In New York State, when we asked people, 48 percent of the children whom we interviewed had worked in fields that were still wet with pesticides despite the fact that there is a reentry time and that that isn't supposed to happen. I think that our numbers are not unusual because, in 1980, similar studies were done in Florida, and people's experience elsewhere has been not dissimilar.

This is a public health problem, and it matters because maybe no one died that we know of, although I think that could be disputed, but we know that people have gotten sick and gone to the hospital. Last November, in Ruskin, Florida, 112 people were exposed in a field which had been sprayed the day before and had not sufficiently dried, and there was a very heavy dew. When they went in there, they got wet, and what they were then coated with was clothing full of wet pesticides.

Eighty-four of those people went to a medical center. Fifty went to a hospital by EMS. Thirteen of them were admitted to a hospital for lengths up to one week, and two of those people ended up in the intensive care unit, one with seizures and one with cardiac arrhythmias. I think that when you know that 48 percent of the children, and there were even more adults who said they had worked in wet fields with pesticides, we are sitting on a situation where that could happen in New York State, in California, in Texas; it could happen anywhere.

We have not had another mass poisoning of the size of Ruskin, but it would seem prudent to do something about it before we do have more people who are injured. The manufacturers do recommend lengthier times, so does the EPA. It is important, I think, that we educate people that these really are hazardous substances. I think a lot of people do believe that the government would not allow you to sell something if it weren't safe, and that sort of upholds the necessity, I think, for a strong regulation.

Chairman **Miller**. You need to wind up your statement soon.

Dr. **Pollack**. Sorry. I just wanted to say that 30 percent of the children also had been sprayed while working in the fields, and one of those children was eating lunch while he was sprayed.

In addition, because migrant farm workers and farmers live adjacent to fields or in the fields, when aerial spraying is done, they also are sprayed. There is a lot of concern about the health effects of that. Notification would help ensure at least that people know and could take some protective measures.

[Prepared statement of Susan H. Pollack, M.D., follows:]

Prepared Statement of Susan H. Pollack, M.D., Instructor, Community Medicine and Pediatrics, Mount Sinai School of Medicine of the City University of New York, and Philip J. Landrigan, M.D., M.Sc., D.I.H., Chairman, Department of Community Medicine and Professor, Department of Pediatrics, Mount Sinai School of Medicine of the City University of New York, New York, NY

Chairman Miller and Members of the Select Committee:

My name is Susan H. Pollack, M.D. I am an instructor in Community Medicine and Pediatrics at the Mount Sinai School of Medicine in New York City. With Dr. Philip J. Landrigan, Chairman of the Department of Community Medicine at Mount Sinai, I have been working on a two year epidemiologic study of the health hazards of child labor. This project is supported by funding from the W.T. Grant Foundation and from the National Institute for Occupational Safety and Health (NIOSH). My curriculum vitae is attached. I am pleased to appear before you today to discuss the exposure of children to occupational and environmental toxins.

Introduction

Children may incur exposure to potentially hazardous chemicals in a variety of ways. These include environmental exposures via contamination of drinking water, contamination of foods, contamination of soils in which children may play, and air pollution. Children may also be exposed to chemicals used in the home and on lawns. Working parents may unknowingly expose children to toxins brought home from their workplaces; lead, asbestos, PCBs and pesticides have all been implicated in "fouling one's own nest". Lastly, children may incur exposures at work.

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Children's Exposures to Toxins in the Home

One of the better known toxins to which children are exposed at home is lead. While flaking lead paint in decaying inner city housing poses a great hazard to many children at levels that were once considered safe, renovation of suburban and rural homes also poses a significant risk, a risk which remains unappreciated by many homeowners, and by those who provide do-it-yourself tips. In the renovation process large amounts of lead dust may be generated by sanding or, even worse, large amounts of lead fumes may be created by heat removal of paint. Dr. Herbert Needleman is here today to address the hazards of lead in depth, so we will direct our testimony to other hazards.

Many homes in America still contain asbestos used to insulate pipes, boilers and beams. While intact asbestos does not pose a health hazard, any situation in which the asbestos becomes friable or frayed allows asbestos fibers to become airborne. It is these breathable fibers that pose health hazards including excess of risk of two forms of cancer -- malignant mesothelioma and lung cancer. It was this hazard that led the EPA and the American Academy of Pediatrics to conclude that

> "A total of approximately 100 to 7,000 premature deaths are anticipated to occur as a result of exposure to prevalent concentrations of asbestos in schools containing friable asbestos materials over the next 30 years. The most reasonable estimate is approximately 1,000 premature deaths. About 90% of these deaths are expected to occur among persons exposed as schoolchildren"

Children may also be exposed to asbestos at home when adults unaware of the hazard disturb intact asbestos in the process of home plumbing work and renovation, or discover fraying asbestos but fail to recognize the hazards. We have recently heard of another manner in which children may be exposed to asbestos: families living in buildings with inadequate heat have stripped asbestos insulation off pipes in order to allow hot pipes to contribute some warmth to their freezing apartments. This removal operation, with families present, generates large amounts of breathable asbestos, and if frayed ends of insulation remain, children may have years of ongoing exposure.

Children's Exposures to Toxins at Work

Children who are employed may be exposed to toxins in the workplace. In 1988, more than 4 million American children worked outside of their homes. For some of these children, the major health hazard of employment is injury related to slicers, tractors and other, often legally proscribed, machinery. But for other children at work toxic exposures are a major health hazard.

Children in garment sweatshops spend long days in poorly ventilated rooms. Shops in which leather work is performed often reek of solvents, glues and dyes, which may be neurotoxic given sufficient exposure over a duration of time.

Some teens work for demolition crews which handle asbestos removal in an unlicensed and unacceptable manner. Ignorant of the risks or nervous about making waves, these children may incur significant exposure to a known human carcinogen. Later in life when they find out more about the potential risks of asbestos exposure, they may come to our clinic to be examined. We can examine them, but we cannot undo the exposure.

Many teens are employed by gasoline stations, where they may spend more than 20 hours per week pumping unleaded gasoline. Unleaded gasoline contains 4 to 5 per cent benzene. Benzene is a proven human carcinogen. Studies of adult workers with benzene exposure have taught us that benzene can cause leukemia and lymphoma in humans. No one has yet published such a case in a child worker, but it would certainly seem prudent to avoid exposure to benzene as much as possible. New technology for recapture of fumes, required by law in states like New Jersey, has done much to decrease the amount of toxic fumes inhaled by gasoline station employees, but skin exposure remains a significant route of hazardous exposure.

Children in a number of occupations have significant exposures to pesticides. Many teenagers work in lawn care and landscaping. While this used to be a job of lawn mowing and bush trimming, with the advent of chemical lawn treatments, exposure to fertilizers and herbicides has become a more common part of lawn care. Adolescents who work for lawn care companies at least know of their exposure, though it is unclear how much personal protective equipment is worn. (Aerial tree spraying is often done without such equipment, despite the fact that pesticide spray mists down onto the appliers.) With the increasing use of chemical lawn treatment, more lawn mowing teens may find themselves working on lawns recently sprayed without knowledge of that exposure.

Migrant farmworker children also incur significant exposure to pesticides, despite federal child labor laws which prohibit hazardous exposures. Children work in the fields for long hours, which increase the duration of exposure. Lack of sanitary facilities with water for hand washing increases the possibility of dermal absorption and ingestion.

Of 50 working migrant farmworker children whom we interviewed from a community in New York State, 4 boys under age 18 stated that they had mixed pesticides, despite the fact that this activity is specifically prohibited as hazardous under federal child labor law. Three boys had applied pesticides. Pesticide manufacturers specify re-entry times after application. No workers should re-enter a field within that time, yet 48% of the children whom we interviewed had worked in fields still wet with pesticides. Pesticides are sprayed either aerially from planes or, in the case of orchards, by tractor. Ideally all workers should be notified of plans to spray fields in which they work. However, 36% of the working children in our survey had been sprayed, either directly or by drift, while working in the fields. One had been sprayed while eating lunch.

Because migrant farmworkers (and farmers) often live adjacent to or surrounded by fields, the spraying of the fields frequently also sprays homes as well. Of the children in our survey, 34% replied that the camps where they lived had been sprayed. Some growers notify residents of spray operation, while others do not. In 1988, a Hudson Valley migrant farmworker mother testified that with such notification, she would have closed windows and taken in laundry.

Without notification, spray would come in her open windows, cover dishes and the kitchen table, and precipitate asthma attacks in her severely asthmatic toddler. In many parts of the country, farm land previously sprayed is being rapidly turned into housing developments, without evaluation of possible pesticide contamination of soil, ground and surface waters nearby.

This is but a brief overview of the many toxic hazards encountered by children in our society, but I hope that it will give you some flavor for the wide range and the potential severity that may be posed to children by these exposures.

I shall be pleased to answer questions.

Chairman MILLER. Dr. Wilkinson.

STATEMENT OF CHRIS F. WILKINSON, Ph.D., MANAGING TOXICOLOGIST, RISKFOCUS, VERSAR INC.

Dr. WILKINSON. Mr. Chairman, members of the committee, I want to thank you today for inviting me to testify. My name is Chris Wilkinson. I am managing toxicologist of RiskFocus Division of Versar Inc., a health risk consulting company in Springfield, Virginia.

Prior to moving to Versar, I was on the faculty of Cornell University for 23 years where I was a professor of insecticide chemistry and toxicology and also director of Cornell's Institute for Environmental Toxicology. The views that I offer today are mine and do not necessarily reflect those of Versar.

Any examination of children's health status is obviously of great importance. Unfortunately, in recent years, the entire issue of potential environmental health hazards has been clouded by sensational media attention and characterized much more by alarmism, fear and confusion rather than legitimate scientific concerns. Unfortunately, the truth and facts of the matter have often been swept aside, and the conventional wisdom on many of the issues is simply wrong.

I am pleased to have this opportunity today to briefly discuss a few issues that may be of importance to your committee. I am going to focus most of my prepared comments on the pesticide issue that, of course, has been very much in the fore in recent years, and also I am going to concentrate particularly on pesticide exposures in the general population as opposed to some of the issues involving occupational exposures.

Risk is simply the probability that some kind of an adverse effect is going to occur. In the case of a chemical, it, of course, is dependent primarily on the nature of the chemical itself, that is, on the toxicity of the chemical, and, of course, on the level of exposure. Exposure is very important.

It is frequently claimed that children are at comparatively greater risk from pesticides than adults because they do in fact ingest more on a percent body weight basis. Certainly, children do eat more fruits and vegetables than adults per unit of body weight. So it is true that they can be expected to ingest somewhat higher amounts of pesticide residues than grown-ups.

Unfortunately, this issue has been exaggerated by a lot of people who claim that children are at serious risk of adverse health effects, whereas the truth is that even under the very worst exposure assumptions—and I emphasize "very worst," children ingest no more than two or three times the level of adults. I should stress that this dose does not begin to approach a level that will cause any adverse health effects that we know of.

What the public and the Congress need to remember in their deliberations is that in this country the levels of pesticides on fruits and vegetables sold commercially are hundreds of times lower than the levels known to elicit any adverse health effects. Up to about 80 percent of our crops contain no detectable residues at harvest, and the vast majority of the remaining fraction contain residues well within legal limits.

When you add that to the fact that you wash the materials, you peel them, you trim them, and so on, it tends to further reduce the already minuscule levels of pesticides prior to actual consumption. Unfortunately, there are those who generate undue public anxiety by claiming that the presence of a pesticide or some other substance in food or water is a deadly danger at any level. Such claims are quite irresponsible.

I think it is perhaps unfortunate in this respect that we have developed in this country exquisitely sensitive, analytical chemical procedures that allow us to go out and measure infinitesimal amounts of chemicals in the environment. This has been used by some political interest groups and advocates to give the false impression that we are literally wallowing around in this sea of deadly chemicals. In my view, this is a gross misrepresentation of the facts.

The assessment of toxicologic risk must be a scientific process. This country cannot afford to have its environmental policies driven by emotion, ignorance, or hysteria because the stakes are just too high. The U.S. regulatory process today demands numbers. That is the way we have it set up. Sadly, the science involved in the risk assessment process has been bent and manipulated to produce those numbers to the point where, in many instances, regulatory policy, in my view, has subsumed scientific risk assessment.

Increasingly, regulatory decisions are made that are based on worst case scenarios and assumptions that are completely unrelated to scientific data. The final result often bears little relationship to reality. I hate to come back to it again, but a good example of this was the 1989 ALAR scare, when public policy in the U.S. was clearly influenced by panic, media hype, consumer advocates, and special interest groups.

I think we should perhaps ask the question why it was that after reviewing exactly the same data base that was available to people here in the U.S., the British concluded that ALAR represented no risk to either children or adults. I certainly hope that the answer to this is not related to the lead residues in the British public that we referred to earlier today.

For the record, I do want to add that, for my sins, I was a member of the Scientific Advisory Panel that first reviewed the ALAR data base for the EPA. I must say that the story that I heard this morning was really quite at odds with the way that I understand it and I experienced it at the time, because that SAP that I worked with was unanimous in the fact that ALAR in fact was very little risk and that the EPA was overstating those risks.

I should add that removal of ALAR from commerce in this country will make not one wit of difference to the health of children or adults in this country. Sound regulatory policy must be developed and implemented pursuant to objective evaluation of all available data and decisions made based on the total weight of scientific evidence available.

As to children specifically, there is neither scientific evidence nor epidemiologic data to support the claim that children are always more sensitive or risk prone than adults to the potential effects of all environmental chemicals. Obviously, there are some situations, we are hearing some this morning, where an infant might be at greater risk to a given toxin, just as there are also cases in which adults or elderly people are more susceptible.

In short, it is really not possible to make blanket conclusions on this issue. Clearly, we must continue to pursue research on these areas and to identify any human subpopulations, children, pregnant women, senior citizens, and so on, who may in fact be at greater risk to certain chemicals. There are undoubtedly various situations where risks associated with some of the chemicals we have talked about are a problem, and they should be identified.

However, at the present time, in the case of pesticides, I want to stress that there are no data to suggest that children are at greater risk than adults. I believe that new or more stringent regulations cannot be justified on unproven and hypothetical conjecture that children, by definition, are always more sensitive to the potential adverse effects of such chemicals.

Thank you for this opportunity to speak to you today.

[Prepared statement of Chris F. Wilkinson, Ph.D., follows:]

Prepared Statement of Chris F. Wilkinson, Ph.D., Managing Toxicologist, RiskFocus, Versar Inc.[1]

MR. CHAIRMAN and OTHER DISTINGUISHED MEMBERS OF THE COMMITTEE. Thank you for inviting me to testify today.

My name is Christopher F. Wilkinson. For the past year and a half I have been Managing Toxicologist with the RiskFocus Division of Versar, Inc. a private health risk consulting company headquartered in Springfield, VA. For 23 years prior to my move to Versar, I was a faculty member of Cornell University, where I was Professor of Insecticide Chemistry and Toxicology and Director of Cornell's Institute for Environmental Toxicology.

The subject that the Committee is addressing in these hearings is, indeed, one of great importance and one that has attracted a great deal of sensational media attention during the last year or two. There has been an enormous increase in public fear and confusion over the potential health risks associated with certain groups of chemicals (e.g. pesticides) that are important parts of our modern technology. There has also been a great deal of misunderstanding on the facts surrounding the issue and the development of mistrust and suspicion of both government and the agricultural chemical industry by a substantial segment of the public.

This is an extremely complex issue and requires a lot of careful evaluation before decisions can be made. Today, I wish to cover just a few points that I feel are of importance to your deliberations.

TOXICOLOGICAL RISK

Risk is defined simply as the probability that an adverse effect of some kind will occur. In the case of a chemical such as a pesticide, the potential risk to human health is a function of the toxicity of the chemical (i.e. its capacity to cause an adverse effect) and the level of exposure. In turn, the toxicity of a chemical will depend in turn on the nature of the chemical itself and on the sensitivity of the exposed individual or subpopulation.

RISK = TOXICITY X EXPOSURE

Since the intrinsic capacity of a chemical to cause an adverse effect will remain essentially constant, the question of whether children might be at relatively greater risk than adults will be determined by:

- the presence of specific physiologic or other factors that might cause a child to be especially sensitive to the chemical;

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[1] The material and opinions contained in this testimony should not be construed in any way as representing the views of RiskFocus or Versar Inc.

- factors that might cause a child to have a particularly high level of exposure to the chemical.

ESTIMATING EXPOSURE

The importance in risk assessment of having a good measure of the level of exposure to a chemical cannot be overstated . Unfortunately, for many,the very fact that a pesticide (or pesticide metabolite) is present in food or water, *at any concentration*, is a cause for immediate concern. It must be realized, however, that such pesticide residues are present in extraordinarily low concentrations, usually measured in parts per million (ppm) or parts per billion (ppb). Few non-scientists have the faintest idea how big, or rather how small, the units really are:

Unit	ppm	ppb
Length:	1" in 16 miles	1" in 16,000 miles
Time:	1 min. in 2 years	1 sec. in 32 years
Money:	1 cent in $10,000	1 cent in $10,000,000
Population:		1 person in China

A few years ago we had great difficulty in measuring 1 ppm of anything. Now we routinely measure ppm and ppb and occasionally we can measure ppt (parts per trillion) and ppq (parts per quadrillion). Our current analytical chemical capabilities are truly amazing and they allow us to find the smallest traces of almost anything we choose to look for. This has tended to heighten public fears about the risks of pesticides in our food and water because it gives many the impression that we are wallowing in a sea of potentially dangerous chemicals. What we must remember is that we no longer live in a pristine environment. If we choose to use pesticides and release them into our environment and our food supply, we must accept the fact that we will always be able to measure trace residues of these materials in our food and water.

During the last few years there have been repeated claims by consumer advocate and environmental groups that levels of pesticide residues in a wide variety of fresh and processed fruit and vegetables are at a dangerously-high level. Such claims, based on a series of worst-case assumptions and scenarios are quite at odds with the facts. The results of all federal (FDA), state and private analyses consistently show that levels of pesticide residues are well below those likely to cause any adverse effects on human health. Indeed, all available data clearly show that the vast majority of crops (60-80%) contain no detectable residues (measured at the farm gate on the raw agricultural commodity) and of those containing measurable residues most are well within legal tolerances; only about 1% are found to be out of compliance and even here there is a vanishingly small probability that they could cause any adverse health effects. It should be emphasized that subsequent processing (peeling, trimming, cooking etc.) tends to lower these residues considerably before the commodity is actually consumed; the extremely small residues present in food as it is actually consumed is clearly shown by the results of the FDA's "total diet studies."

It is important to recognize that, as the level of exposure to any chemical decreases, the intensity of any toxicologic effect also decreases. Eventually, a level of exposure is reached, the so-called threshold, below which no effect will be observed. *The levels of pesticide residues in fruit and vegetables are hundreds of times lower than those likely to elicit any adverse health effects.*

While there is no question that we should continue to be vigilant and, wherever possible, strive to improve the surveillance of our food supply for pesticide and other residues of synthetic chemicals, it is my opinion that our current level of analysis is adequate to provide the information we need. I do not believe that doubling or tripling our current analytical capabilities (at enormous cost) would provide a great deal more information than is already available.

Exposure of children to pesticide residues in food.

In general, adults and children eat the same fruit and vegetables as adults. However, it is important to recognize that a child's diet is different from that of an adult both in type and amount and, per unit of body weight, children eat more fruit and vegetables than adults. Consequently, it is true that, per unit of body weight (e.g. intake per kg body weight) children can be expected to ingest a greater amount of pesticide residues. This factor has been highly sensationalized and exaggerated by some and has been used to indicate that many children are at serious risk of suffering an adverse health impact. It must be recognized that, under a worst-case scenario, children ingest no more than two or three times the level of adults (per kg body weight) and that with existing levels of safety these small differences do not begin to approach a what can be considered a hazardous level.

One area here that does require improvement is more precise information base on the dietary characteristics of infants and children. One would imagine that the question of exactly what infants and children eat and how much would be relatively easy to answer; in fact little reliable information is available.

TOXICOLOGY RISK ASSESSMENT

The assessment of toxicologic risk should firstly and foremost be a scientific process. While the science of toxicology is rapidly advancing our state of knowledge and understanding of how chemicals exert their toxic effects, it must be emphasized that evaluating human health risks will always be a highly uncertain process. There are many problems inherent in attempting to predict likely effects in humans exposed to traces of a given chemical in food or water from the results obtained in rats fed truly massive daily doses for a lifetime.

In the last few years we have tended to misuse and abuse science in order to develop and conduct regulatory policy. The regulatory process demands numbers (health guidelines, cancer risk estimates etc.) and the science involved in the risk assessment process has been stretched, bent and manipulated to produce such numbers. Gaps in scientific knowledge have been obviated and "resolved" by making a variety of conservative assumptions and regulatory guidelines. Regulatory policy has intruded into the scientific aspects of risk assessment and, in many cases, the two have become indistinguishable. Certainly they cannot readily be distinguished by nonscientists and the result has been a kind of public "brain-washing" that, indeed, we know a lot more about toxicology than we really do and have very precise methodology for evaluating human toxicological risks. We do not.

Because of the natural desire of regulators to remain firmly on the side of prudence, the risk estimates that are developed are typically based on a series of worst-case scenarios and highly conservative policy assumptions. The final result is often so conservative and hypothetical that it has little or no relationship with the real world. There is also little doubt that many of the important regulatory decisions being made today are based on factors completely unrelated to science. A good example of this was the Alar saga of 1989. After reviewing precisely the same database as that considered by the EPA, the British conclusion on this matter was that even the highest Alar residues in food constituted essentially no risk to either adults or children. There is no question that regulatory action in this country is often strongly influenced by public opinion and Alar is a prime example of this.

This is the primary reason why it is so important to improve the communication process and raise the public's understanding of chemical and other risks so that issues of this type can be placed in better perspective. Only then will the public be able to play a more effective role in the regulatory process.

We must strive to improve the level of scientific input into our regulatory decisions. *Sound regulatory policy can only be developed and conducted on the basis of a complete, objective evaluation of all the available data and decisions must be based on the total weight of evidence available.* Clearly, informed subjective value judgements are necessary but unfounded emotional hype should not play a role in the process.

Are children inherently more sensitive to the adverse effects of pesticides and other chemicals ?

A conventional wisdom has developed that children are always more sensitive than adults to the action of pesticides and other synthetic chemicals. This has become a highly emotional issue that has both angered and frightened people. But where is the evidence to support this view.

At the present time there is neither scientific evidence nor epidemiologic data to support the view that children are always more sensitive than adults to the effect of environmental chemicals. While certainly there are some situations in which infants might be at greater risk to a given toxicant, there are others in which adults or older people are more susceptible. Few, if any, generalizations can be made; each situation must be evaluated on its own merits.

The possibility that certain human subpopulations (children, senior citizens, pregnant women etc.) might be more sensitive and consequently at greater risk from certain chemicals is certainly something that should be carefully considered and evaluated and it is true that, in the past, we have not given as much attention to this as perhaps we should. As a result we don't have as much information as we would like to have and this tends to frighten people. There are many foretellers of doom who expound the philosophy that "if only we conducted the appropriate tests" we would be sure to uncover a serious problem. On the other hand, there is nothing to suggest that any problem exists.

While more information is always advantageous, it should be emphasized that data on child sensitivity is extremely difficult to obtain. Extrapolation from the effects of chemicals on immature rodents or other animals is of questionable value and epidemiologic studies with groups of children are equally difficult to conduct and interpret. In the case of pesticide residues, there are no data to suggest that any problem exists. *It is difficult to justify developing new and more stringent regulatory policy on the unfounded assumption that children are more sensitive to the potentially adverse effect of these and other environmental chemicals.*

THE NEED TO ESTABLISH REGULATORY PRIORITIES

As a nation we have limited resources to identify and resolve technological risk. Consequently, we must be extremely careful to establish our priorities so that attention and precious resources are not squandered on trivial unproductive issues that have no impact in reducing risk or improving public health. All resources that are funneled into one area automatically mean that there is less for resolving some other possibly more important issue. *I am concerned that many of our regulatory priorities are currently being dictated by the emotional, non-scientific claims and demands of a few highly vocal individuals and organizations.* In my opinion, these individuals are do a serious disservice to society by causing needless fear and concern among a large segment of the public and placing enormous pressure for immediate (often unnecessary) "action" on state and federal legislators and regulators.

Chairman MILLER. Mr. Feldman.

STATEMENT OF JAY FELDMAN, NATIONAL COORDINATOR, NATIONAL COALITION AGAINST THE MISUSE OF PESTICIDES, WASHINGTON, DC

Mr. FELDMAN. Good afternoon. Thank you for the opportunity to speak.

I am Jay Feldman, national coordinator of the National Coalition Against the Misuse of Pesticides. I would like to note that our staff toxicologist, Katherine Carr, played a very important role in assembling the information in the written testimony.

There is no dispute that the volume of use of pesticides is huge in this country. We are talking about 2.6 billion pounds of insecticides, fungicides, rodenticides, antimicrobial household pesticides, as well as wood preservatives. The exposure scenarios are great for children as well as the general population.

We have talked a lot today about food use exposures. NAS looked at just 28 pesticides of over 70 carcinogenic pesticides and found them at tolerance to be responsible for potentially over a million cases of cancer. Contaminated air has been looked at by a recent national nonoccupational pesticide exposure study by EPA, which found at least five pesticides in indoor ambient airs at levels 10 times greater than outdoor air.

Another study looked at chlorpyrifos, which is a widely-used insecticide used around our homes and in this building, perhaps, for cockroach control, most notably, applied at normal levels, found substantially higher concentrations at the infant breathing zone compared to the adult breathing zone, which is closer to floor.

Ground water and drinking water, 77 pesticides found in the ground water of 39 states. Then lawns, and playfields, and schools, of the 33 most commonly used pesticides on lawns, 9 cause cancer, 10 cause birth defects, 3 cause reproductive effects, 9 cause liver and kidney damage, and 20 cause neurological effects or are known to affect the nervous system. The point is, within these exposure scenarios, we have behavioral effects which put children at significant and unique risk.

Moving on to the question of specific adverse effects to children, which I was going to go over until the previous testimony, it is very important to point out that we are not just looking at a question of how much is ingested, although, yes, that is a major element of the discussion; we are looking at the unique qualities of children that put them at substantial risk, which was not addressed by the previous testimony.

Because growing children are more active than adults, they require more food and oxygen, a higher dose of exposure. But more importantly or as important, we are looking at questions of barriers to absorption of toxic substances which are not well developed: the blood brain barrier; skin absorption has been found, for instance with chlorpyrifos, to be much higher in laboratory studies in young animals rather than or compared to adult animals.

Children are less equipped to manage toxic exposure. Kidney and liver organs, which, as you may know, are detoxification organs to excrete foreign substances, are incompletely developed at birth.

Immune systems are not as well developed. Neurotoxic effects simply are greater in children.

The NAS looked at the issue of mental health and found that 12 percent of 63 million children in the U.S. suffer one or more mental disorders, and it did identify toxic substances as contributing to these risk factors.

Organophosphates, which are one of the most commonly used class of pesticides in the United States, do undergo neurotoxic testing but only for limited screens to assess delayed paralytic reaction following high exposure.

The susceptibility to cancer-causing effects of pesticides or carcinogens is much greater in children than adults. Again, not a question of volume of ingestion but vulnerability of the population.

The National Cancer Institute conducted a study in which they looked—an epidemiological study, I should say, in which they looked at household and garden pesticides and correlated that with home use and found that childhood leukemia represented a sevenfold increase in those households compared to households where garden and household pesticides were not used. Between 1950 and 1986, the last year for which good data is available, the incidence of childhood cancer increased 21.5 percent.

There is no question that farm worker children are the first in line. I conducted a series of hearings in Florida, Texas, and California in 1979 and 1980 for the EPA, and there is no question that the lack of control is just rampant. It is not uncommon for bell peppers having just been sprayed that morning to be cut open and used as cups by children who are waiting in cars on the edge of the fields for the parents to go through the day's work.

Toxic sensitization is increasing. The question of chemical sensitivity is one that has to be looked at.

Let's quickly look at the regulatory system. We have talked about this question of exposure, volume of use, vulnerability on the part of children, what is the regulatory system doing or not doing? The point is, EPA is not looking at these exposure scenarios.

We have in our office, which I would like to submit for the record, a 1990 internal memorandum in the EPA Toxic Exposure Branch, in which the astounded reviewer, in a memo to his supervisor, asked, "Am I to assume all residential exposure, including all indoor and lawn pesticide uses, are excluded under reregistration until it is completed in nine years?" implying that the answer was yes and that his instructions were not to proceed with those kinds of analyses.

GAO reported that EPA usually develops separate cancer risk estimates only for the U.S. population overall. In an EPA lawn memorandum in 1989, again, reviewers were astounded that EPA failed to collect information on residue on lawns and landscapes and dermal absorption so as to be unable to adequately assess the dose issue that the previous witness raised.

Negligible risk policy has been embraced by EPA and adopted as of 1988, in which EPA wholesale neglects implementation of the Delaney clause of the federal Food, Drug and Cosmetic Act, Section 409.

The point is, EPA is not only dismissing the vulnerability of children to carcinogens, but it is not looking at multiple exposure. If we are looking at the fact that we register 12 carcinogens in the production of pesticides in the production of citrus, then we should be looking at multiple aggregate exposure. On top of that, we need to look at nondietary exposure and ultimately look at the toxic body burden.

Finally, again, a regulatory and statutory problem of working with a risk/benefit statute. The risk/benefit statute assumes that we need to introduce toxic materials into the environment because they yield benefits to society at large. That is how it is described. In effect, what the law is doing is weighing human health against benefits to individual economic interests, not national economic interests.

Therefore, it is incredibly essential to us in the public and to you, we believe, in Congress, to look at whether the assumptions associated with and benefits of pesticides are in fact true. The fact of the matter is, we are showing increasing insect resistance, weed resistance; 447 insect species are showing resistance. We lose 30 percent of the value of crop production to disease.

The current methods of assessing benefits and the implied assumptions brought to that analysis, which, in effect, in the pesticide area justifies this hearing today are simply unfounded. There is no benefit in the area of agriculture for many of our crops to introduce toxic materials when least toxic or nontoxic methods exist. Similarly, there is no benefit to introducing pesticides in a lawn when cultural practices can be used to achieve the same end goal.

There are a number of suggestions we have in our testimony to change and protect us in this regard, maintaining state authority to exceed federal standards is one that we urge you to zero in on, as you may have in the past.

Thank you.

[Prepared statement of Jay Feldman follows:]

PREPARED STATEMENT OF JAY FELDMAN, NATIONAL COORDINATOR, NATIONAL COALITION AGAINST THE MISUSE OF PESTICIDES, WASHINGTON, DC

Mr. Chairman and members of the Committee. Thank you for the opportunity to address you today on a problem of extreme importance to the public and our membership --environmental toxins and their effects on children.

I am Jay Feldman, National Coordinator of the National Coalition Against the Misuse of Pesticides (NCAMP). NCAMP, founded in 1981, has been working with local groups and people across the United States and around the world in an effort to ensure safety from pesticides in our food, water, land and air. Our membership, including those living in urban and rural areas, on and off the farm, is composed of people, including approximately 200 community-based groups in nearly every state, with concerns about a range of pesticide-related issues who seek to reduce and where possible eliminate pesticide exposure while promoting alternative methods of pest control which do not rely on toxins.

NCAMP focuses on the threat of one major group of toxins, pesticides. The huge volume of pesticides used annually for agricultural and nonagricultural use, which the U.S. Environmental Protection Agency (EPA) estimates at 2.6 billion pounds in 1988, raises serious questions about public exposure to known toxins. Children are among the most vulnerable population groups of those exposed. Taken together, this would suggest the need for special attention --with children in mind-- to pesticide use patterns, residues following use, protective risk calculations and the availability of alternative methods of pest control. And yet, nothing approaching this is a part of the regulatory review and restrictions governing the use of poisons that are purposefully added to the environment in homes, schools, parks, along rights-of-way, and on farms. While children occupy a very special place in our culture, they do not occupy a special place in our environmental health policies. While children are special to us all, they are virtually ignored in the setting of pesticide policy.

The driving force behind unprotective pesticide regulatory actions is a risk-benefit standard in the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) which weighs human life against benefits to individual economic interests. And in weighing human life, the young lives are not protected. This terrible inequity and injustice persists under a regulatory system that fails to consider, with any critical analysis, the availability of alternative non-toxic or least toxic methods of pest control. A critical analysis in this area, if implemented by the Environmental Protection Agency (EPA), would find that there is no benefit to poisoning children through the use of lawn care pesticides, for example, when alternative methods of control exist or aesthetic goals are being pursued. A policy such as this would ultimately protect vulnerable groups, such as children. Instead, the existing regulatory system invites the development and sale of more and more toxic products without encouraging or forcing the transition to alternative methods of pest management.

While this proceeds, the scientific community and general public are increasingly aware of the repercussions to public health and environmental quality that result from pesticide use in agriculture, forestry, and urban environments. The special vulnerability of young people to pesticide exposure is easily recognized by virtue of their behavioral and lifestyle differences as well as physiological factors associated with age. In response, health professionals, community-based organizations, school administrators, day care center workers, and others with an interest in protecting children are questioning traditional practices of pest control and supporting alternative methods that do not pose unnecessary risks. In poll after poll, people are saying that they do not want to be exposed to pesticides.

I. Children Are Particularly Vulnerable to Pesticides

Pesticides are poisons by nature and threaten health with immediate or acute toxic effects when exposure is relatively high. Acute symptoms of poisoning include dizziness, nausea, headaches, rashes, sensitization, and mental disorientation. In addition, low level exposure over a period of time may result in chronic health effects. Existing literature links pesticides to a range of chronic health effects including cancer, birth defects, genetic damage, neurological, psychological, and behavioral effects, blood disorders, reproductive effects, and abnormalities in liver, kidney and immune system function.

In addition to their active ingredients designed to kill some target pests, pesticide products contain inert ingredients which are not listed on the label and include everything else, such as contaminants, emulsifiers, solvents, preservatives, and anti-volatility agents. Information on the effects of pesticidal active ingredients is required by law and available to the public. However, a pesticide formulation may consist almost entirely of inert ingredients, yet these are considered trade secret information and the public is not allowed to know. Of the 1200 inerts, EPA knows 55 to be of "toxicological concern" because they have been shown to cause cancer, nerve damage, adverse reproductive effects, or other chronic effects. In some cases the inert ingredients may be more toxic than the active ingredient in a pesticide formulation. EPA does not have adequate data to assess the toxicity of 700-800 of the inerts and regards about 275 as innocuous.

No one knows how many Americans are acutely poisoned by pesticides each year because there is no centralized, nationwide program or policy to collect this information. However, statistics available from a variety of sources indicate the number of poisonings is significant, especially for children. Of the 63,345 pesticide exposures reported to Poison Control Centers in 1988, 38,002 or 60% represented children under age 6.[1] A Consumer Product Safety Commission survey of emergency room admission in 1985 found pesticides to be the most frequent cause of poisoning in young children, following medicines.[2]

II. Pesticide Exposure to Children is Varied and Widespread

We are experiencing a national pesticide exposure dilemma. As the number of surveys multiplies, the severity of the problem is disturbingly apparent. While purposefully introduced into the environment for an intended target, these poisons insidiously find their way into our children's homes, playgrounds, playing fields, water supplies, schools, day care centers and food. Pesticide residues in food and the ingestion of these chemicals represents a serious concern which has been well documented. In fact, EPA has called it one of the three most serious public health threats, next to worker exposure to chemicals and radon.[3]

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[1] Litovitz, T.L. et al. 1988 Annual Report of the American Association of Poison Control Centers National Data Collection System. *American Journal of Emergency Medicine* 7:495-545, 1989.

[2] *EPA Journal*, May 1987, p. 27.

[3] U.S. Environmental Protection Agency, *Unfinished Business: A Comparative Assessment of Environmental Problems*, Washington, D.C., 1987.

Non-food related pesticide exposure through indoor and outdoor air, surfaces and landscapes are very real for children. A four-year old Michigan boy spent five months in the hospital last year recovering from severe mercury poisoning. His parents had painted the interior of their home with a paint containing a mercuric fungicide and his exposure to the mercury vapors rendered him unable to walk, irritable, and caused his skin to slough off after a painful rash.[4] In 1980, a California infant was rushed to the hospital where he suffered respiratory arrest. Organophosphate poisoning was suspected and later confirmed even though his parents had kept him away from home during termite and roach spraying soon after his birth.[5] California Department of Food and Agriculture sampling found chlorpyrifos (Dursban™) on dish towels, food preparation surfaces, and the infant's clothing.

The important contribution to total pesticide exposure that may be attributed to contaminated house dust, air, soil, surfaces, and water has not enjoyed the same media and regulatory attention as food residues. It is vital to realize that the dangers of pesticides go beyond food issues and, in fact, exposure through other media may be far greater and threatening.

We will summarize the range of exposure concerns as they affect children:

A. Food Issues Trigger Concern

Pesticide residues in food have attracted important public intention. Beginning in 1983 with public attention focused on ethylene dibromide (EDB) (a cancer causing grain fumigant) contamination of grain-based foodstuffs, public concern of pesticide residues surged. Food industry polls in that year showed a dramatic shift in public concern with pesticide residues in food, ranking it ahead of traditional food safety issues, such as colorings, dyes, salt, cholesterol, etc. More recently, daminozide (Alar)™ residues in apples and apple products raised public concern yet again. Citing the high cancer risk to children and infants who consume apples and apple products much more than adults, the Natural Resources Defense Council and other interested parties petitioned U.S. EPA in 1986 to ban the use of Alar on food,[6] which expedited an otherwise unresponsive regulatory process, leading to marketplace pressure and voluntary cancellation by the manufacturer for food uses.[7]

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[4] Center for Disease Control, "Mercury Exposure from Interior Latex Paint - Michigan," *Morbidity and Mortality Weekly Report* 39(8):125-126 (1990).

[5] Center for Disease Control, "Pesticide Poisoning in an Infant - California," *Morbidity and Mortality Weekly Report* 29(22) (1980).

[6] Natural Resources Defense Council July 2, 1986 Press Release: Groups File Petition to Bar Cancer-Causing Pesticide from Food.

B. Studies Indicate Contaminated Indoor Air Represents A Significant Source Of Exposure.

EPA's Non-Occupational Pesticide Exposure Study or NOPES, released in January, 1990, found in the majority of households sampled at least five pesticides in indoor ambient air at levels often ten times greater than levels measured in outdoor air.[8] While NOPES set out to determine household pesticide exposure through air, drinking water, food and dermal contact, the efforts to evaluate exposure via routes other than air were much less comprehensive and inconclusive. However, NOPES does suggest that house dust may be an important source of exposure especially for infants and toddlers, noting potential dermal contact, ingestion, and inhalation of suspended particles. In addition, a study published by Fenske, et al. in the *American Journal of Public Health* in June, examined health risks associated with pesticide residues in air and on surfaces. Chlorpyrifos, a commonly used organophosphate insecticide, was applied according to normal broadcast application techniques.[9] Substantially higher chlorpyrifos concentrations were measured in the infant breathing zone, which is closer to the floor compared to the adult breathing zone. Moreover, ventilation had far less impact on decreasing infant breathing zone levels than adult breathing zone levels. The study concluded that exposures to organophosphate insecticides "following properly conducted broadcast applications could result in doses at or above the threshold of toxicological response in infants, and should be minimized through appropriate regulatory policy and public education."

C. Groundwater and Drinking Water Add to Exposure.

Dr. George Hallberg with the Iowa Department of Natural Resources has published widely on groundwater pollution and remarks "pesticides are leaching through the soil and into groundwater far more commonly than the preconceptions of a decade ago would have predicted."[10] EPA's Office of Pesticide Programs Environmental Fate & Groundwater Protection Branch cites the detection of 77 pesticides in 39 states in their most recent report.[11] Results of the National Pesticide Survey, an EPA effort to survey a representative sample of drinking water wells nationwide for pesticides and nitrates, will not be released officially until this Fall. However, preliminary results indicate widespread detections of nitrate/nitrite contamination.[12] The toxic effect of nitrites is methemoglobinemia. Infants are particularly susceptible to nitrate-induced methemoglobinemia, also known as blue baby syndrome, because of their low acid production, large numbers of nitrate-reducing bacteria and the relatively easy oxidation of fetal hemoglobin.[13] Iowa's 1988-89 state-wide rural well-water survey reports that 18.3% of private, rural wells contain nitrate concentrations exceeding recommended health advisory levels.[14]

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[7] 54 FR 47492 November 14, 1989.

[8] U.S.-EPA. *Nonoccupational Pesticide Exposure Study* (NOPES). EPA/600/3-90/003, January 1990. Atmospheric Research and Exposure Assessment Laboratory, Research Triangle Park, North Carolina.

[9] Fenske, R.A. et al. (1990). "Potential Exposure and Health Risks of Infants following Indoor Residential Pesticide Applications." *American Journal of Public Health* 80(6):689-693.

D. Children May Encounter Dangerous Levels Of Pesticide Contamination On Lawns, Playfields, And At School.

The U.S. General Accounting Office (GAO) estimates 67 million pounds of pesticidal active ingredients are applied to U.S. lawns annually.[15] *Urban Pest Management*, a National Academy of Sciences report, suggests that "suburban lawns and gardens receive heavier pesticide applications than most other land areas in the United States".[16] In Phoenix, the Department of Health Services received a rash of complaints from children experiencing rashes, headaches, and even a swollen face as a result of pesticide use at the school.[17] In many cases students had been playing on pesticide treated fields.

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[10] Hallberg, G.R. (1989). "Pesticide Pollution of Groundwater in the Humid United States". *Agriculture, Ecosystems, and Environment* 26:299-367. Elsevier Science Publishers, Amsterdam.

[11] U.S.-EPA. *Pesticides in Ground Water Data Base 1988 Interim Report.* December, 1988. Office of Pesticide Programs.

[12] U.S.-EPA. Press Advisory September 1, 1989. "EPA Releases Interim Results of National Survey of Pesticides in Drinking-Water Wells." Office of Public Affairs, Washington, DC.

[13] Ellenhorn, M.J. & D.G. Barceloux. (1988). *Medical Toxicology*, Elsevier, New York, p. 844.

[14] Iowa State Department of Natural Resources. "Iowa State-Wide Rural Well-Water Survey 1988-89". Des Moines, IA.

[15] U.S. General Accounting Office (GAO). *Lawn Care Pesticides: Risks Remain Uncertain While Prohibited Safety Claims Continue.* Washington, D.C. GAO/RCED-90-134. March, 1990.

[16] National Academy of Sciences (NAS). (1980). *Urban Pest Management.*

While statistics on poisoning in schools are not available, NCAMP receives numerous inquires from school administrators, teachers, and parents who are concerned over the use of pesticides in their schools. Many of these requests are from teachers or parents with children who believe their health has been affected by pesticides used in or around schools. Schools frequently use chemical treatments on a regular basis regardless of whether or not the pest is present and applications are often done by untrained individuals. In May 1989, for example, a West Virginia junior high school was closed by the National Institute of Occupational Safety and Health after concentrations of chlordane eleven times the evacuation threshold were found. The chlordane had been misapplied by the school janitor.[18] Two employees of a YMCA day care center in Massachusetts have challenged the Y's policy of weekly spraying of organophosphate insecticides for cockroach control.[19] The insecticides used, chlorpyrifos and propetamphos, are normally applied every one to three months.

E. Behavioral Differences Increase a Child's Likelihood of Attaining a Toxic Exposure.

Children are much more likely to crawl around places treated by pesticides or to roll on turf or climb through shrubbery that has been sprayed. They are also more likely to put things into their mouth. Furthermore, Fenske's work mentioned above indicates that pesticide concentrations are higher in the breathing zone of infants and children.

III. Pesticides Present Specific Adverse Effects in Children

A variety of age-related physiological factors explain the increased sensitivity that children face. Not only do younger and smaller people by nature receive a higher dose of toxins, they have a decreased ability to eliminate toxins and their target organs may be more sensitive to toxic effects.

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[17] Vrcan, L. "Are Pesticides a Cause for Concern?" *School and College Product News*. February, 1987.

[18] National Coalition Against the Misuse of Pesticides. *Technical Report*. May, 1989.

[19] "Y fires pair who fought toxin use at day-care center." June 20, 1990. *The Patriot Ledger*. Quincy, MA.

In addition, the probability of an effect such as cancer, which requires a period of time to develop after exposure, is enhanced if exposure occurs early in life.

1. **Because they are growing and more active than adults, children require more food and oxygen. Thus, they receive a higher dose of toxins per pound of body weight.**

At one toxic waste site, the difference in exposure between an infant and an adult was calculated. The 15 pound baby had an exposure to contaminated well water 5 times greater than a 130 pound adult in the same family. The calculation took into account the greater consumptions of water and the absorption from bathing or showering but not the increased food or air consumptions by kids.[20]

2. **Young people also receive higher doses because the barriers to absorption of toxic substances are not well-developed in infants and children.**

Their gastro-intestinal tract is more permeable and pinocytosis, the process by which cells actively transport a compound across cell membranes, is increased.[21] For example, upon review of the scientific literature, The Natural Resources Defense Council found that young humans as well as all other species studied, absorb toxic metals such as lead and mercury with significantly greater efficiency than adults.[22] Adults absorb approximately 10% of an oral dose of lead, a child absorbs 40%.[23] Similarly the blood-brain barrier, which serves to protect the adult brain from toxic exposures, is not fully developed at birth.[24] Furthermore, absorption across the skin may be more efficient in young people. Studies in infant rats have demonstrated increased skin absorption of chlorpyrifos relative to adults.[25] Although the stratum corneum, or outermost skin barrier of the infant is considered to be fully developed, the permeability of infant skin has not been measured.[26] However, unexpected fatalities among children treated for lice with malathion or lindane, have been attributed to increased permeability of children's skin.[27] Also, common skin conditions experienced by kids such as rashes and dryness, are known to reduce the barrier properties of skin.[28]

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[20] Harris, R.H. et al. (1984). "Adverse Health Effects at a Tennessee Hazardous Waste Disposal Site." *Hazardous Waste* 1:183-204.

[21] Calabrese, E.J. (1986). *Age and Susceptibility to Toxic Substances*. John Wiley & Sons.

[22] Natural Resources Defense Council (NRDC). *Intolerable Risk: Pesticides in Our Children's Food*. February, 1989.

[23] Goyer, R.A. "Toxic Effects of Metals" in *Casarett and Doull's Toxicology: The Basic Science of Poisons*. Macmillan Publishing Co. 1986. p.598.

[24] Klaassen, C.D. "Distribution, Excretion, and Absorption" in *Casarett & Doull's Toxicology: The Basic Science of Poisons*. Macmillan Publishing Co. 1986. p. 43.

3. Young people are less well-equipped to manage toxic exposures once they occur.

Both the kidney and liver, which function in the detoxification and excretion of foreign substances, are incompletely developed at birth. This contributes to the increased toxicity of substances to newborns.[29] The young are also less capable of protein binding, another important detoxification mechanism. Protein binding functions by segregating foreign chemicals, making them less likely to reach their site of toxic action. A recent review reports that for 30 drugs tested, human infants almost always displayed less of the drug bound to plasma protein than young or middle-aged adults.[30]

4. Finally, some target organ systems, particularly in the very young child, are immature and thus more susceptible to toxic insults compared to adults.

The child's immune system is immature for the first two years of life. Many pesticides affect the immune system and damage there can result in increased problems with allergies, asthma and hypersensitivity to chemicals and a reduced ability to combat infections and cancer. In a community exposed to contaminated drinking water in Battle Creek, Michigan, white blood cells, which are key cells in the immune system, were abnormally low in 8% of adults, and 37% of children.[31] Also, a child's reproductive system does not mature until puberty. DBCP, a soil fumigant pesticide, was cancelled because it was shown to cause sterility in adult male workers.[32] Studies in rats show that it take only 1/8th the dose to cause sterility in a rat going through puberty as it does to cause sterility in an adult rat. A young man in California, exposed to DBCP in well water as a child, found at age 21 that he was sterile and that the damage to his testes looked like typical DBCP damage.[33]

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[25] Shah, P.V. et al. (1987). "Comparison of the penetration of 14 pesticides through the skin of young and adult rats." *Journal of Toxicology and Environmental Health* 21:353-366.

[26] Wester, R.C. et al. (1987). "In vivo percutaneous absorption" in *Dermatotoxicology*, Third Edition. Hemisphere, Washington, DC.Shah, P.V. et al. (1987). "Comparison of the penetration of 14 pesticides through the skin of young and adult rats." *Journal of Toxicology and Environmental Health* 21:353-366.

[27] A. Bainova. (1981). "Dermal Absorption of Pesticides" in *Toxicology of Pesticides*. World Health Organization, Copenhagen. p. 41.

[28] Wester, R.C.

[29] Klaassen, C.D., pp. 49-51.

[30] NRDC.

A. Neurotoxicity, Or Effects On The Brain And Nervous System, Are Greater in Children.

The young have been shown to be at great risk to the neurotoxic effects of a number of toxic substances including pesticides. Of the 31 neurotoxic metals, pesticides, and other organics analyzed by Calabrese in *Age and Susceptibility to Toxic Substances*, there was an age-related difference in susceptibility for all but two.[34] In 66% of the cases with age differences, the young were more susceptible. Many of pesticides in use are neurotoxins. Neurotoxic pesticide effects can range from headaches, dizziness and memory loss to learning disabilities, hyperactivity, seizures, numbness in the hands and feet, and permanent brain damage.

The developing nervous system is particularly vulnerable to neurotoxins for several reasons. As mentioned previously, the blood-brain barrier is not fully developed. In addition, myelination, the process by which nerves are coated with a fat-like substance called myelin, progresses most rapidly in the first two years but is not complete until adolescence.[35] Also, developing neurotransmitter and hormone cell receptors in the brain are vulnerable to disruption by neurotoxic agents. For example, exposure to the insecticide endosulfan when neurotransmitter receptors are forming, affects the number of receptors and their functional maturation.[36] Hormone exposure can also disrupt cell receptor development because certain receptors require reinforcement by the appropriate hormone at a critical stage in development. The chlorinated hydrocarbon pesticides such as chlordane, have estrogen-like properties and can disrupt receptor development.[37]

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[31] Paigen, B. (1986). "Children and Toxic Chemicals." *Journal of Pesticide Reform*. Summer 1986.

[32] NAS, p. 128.

[33] Paigen, B.

[34] Calabrese, E.J.

[35] NRDC.

Childhood is a period of learning. In a few short years, a child must learn the skills necessary to function in adult life. Children must develop motor skills, learn to speak, read and write, and master socially acceptable behavior. The National Academy of Sciences recently reported that 12% of the 63 million children under the age of 18 in the U.S. suffer from one or more mental disorders. It identified exposure to toxic substances before or after birth as one of several risk factors that appear to make certain children vulnerable to these disorders.[38] Currently, federal regulations do not require than any pesticide be evaluated for the effects of low-level exposure on behavior, including such processes as learning ability, activity level and memory, or on emotion, sight and hearing. Organophosphates are the only class of pesticides that are required by EPA to undergo any neurotoxicity testing at all and this is limited solely to a screen to assess delayed paralytic reactions following high level exposure.

The organophosphate and carbamate insecticides are the most neurotoxic classes of pesticides used in the U.S. and are the most common cause of poisoning in agriculture.[39] The organophosphates represent 40% of pesticide registrations.[40] The primary toxic effects of these pesticides is disruption of normal nerve transmission. Studies have found that the young are especially susceptible to the acute effects of the organophosphate insecticides. In fact, young rats are more susceptible than adults to the acute effects of 15 out of 16 organophosphate insecticides tested.[41] For parathion and methyl parathion, the lethal dose is 6-8 times lower than in adult rats.[42] For some organophosphates, the lethal dose in immature animals has been reported to be only 1% of the lethal dose in adults.[43]

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[36] Seth, P.K. et al. (1986). "Neurotoxicity of Endosulfan in Young and Adult Rats." *Neurotoxicology* 7(2):623-636.

[37] NRDC.

[38] National Academy of Sciences. Toxicity Testing: *Strategies to Determine Needs and Priorities*. National Academy Press, Washington, DC. 1989.

[39] U.S. Congress, Office of Technology Assessment. *Neurotoxicity: Identifying and Controlling Poisons of the Nervous System*. OTA-BA-436. Government Printing Office, Washington, DC. April, 1990.

[40] Ibid. p. 49.

[41] Cabrese, E.J.

B. Susceptibility To The Cancer-Causing Pesticide Effects Increase.

Many toxins, particularly carcinogens, damage rapidly dividing cells more than they damage cells that are in a resting state. Children are actively growing, their cells are dividing at a rapid rate, and this makes them more susceptible to toxins such as carcinogens.[44]

Numerous studies have found that exposure early in life puts animals at greater risk of developing cancer than exposure in later life. Enhanced susceptibility is seen even when total exposure is considerably less for the younger animal. This is observed for both direct and activated carcinogens. These and other findings have led researchers to conclude that infancy has "proved to be the most susceptible period to carcinogenesis"[45]

Children rarely fall prey to cancer, yet there are few more tragic events than cancer striking a young person. Between 1950 and 1986 (the last year for which good data are available), the incidence of childhood cancer increased 21.5%.[46] Epidemiological studies of childhood cancer lend very disturbing evidence that exposure to pesticides at home may be an important risk factor. Childhood tumors and blood disorders have been linked with substantial prenatal or environmental exposure to specific insecticides by Infante, et al.[47] while Gold, et al. report that children with brain cancer are more likely than normal controls to have been exposed to insecticides in the home.[48] A study sponsored by the National Cancer Institute indicates that household and garden pesticide use can increase the risk of childhood leukemia as much as seven-fold.[49] The most common exposure of 15 children whom reported to a blood dyscrasia clinic in California stricken with aplastic anemia or leukemia, was found to be exposure to household use pesticides, particularly DDVP and propoxur, a major residential use pesticide.[50]

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[42] NRDC.

[43] Spyker, J.M. and D.L.Avery. (1977). "Neurobehavioral Effects of Prenatal Exposure to the Organophosphate Diazinon in Mice." *Journal of Toxicology and Environmental Health* 3:989-1002.

[44] Paigen, B.

[45] Vesselinovitch, S.D., et al. (1979). "Neoplastic Response of Mouse Tissues During Perinatal Age Periods and Its Significance in Chemical Carcinogenesis." *Perinatal Carcinogenesis*. National Cancer Institute Monograph 51.

[46] Bazell, R. "Cancer Warp." *The New Republic*. December 12, 1989.

[47] Infante, P.F. et al. (1975). "Blood Dyscrasias and Childhood Tumors and Exposure to Chlordane and Heptachlor." *Scandinavian Journal of Work and Environmental Health* 4:137-150.

Early exposure does not necessarily mean early manifestation of cancer. In reviewing the cancer literature, The Natural Resources Defense Council found that "exposure to carcinogens in infancy and early childhood does not necessarily mean that cancer will result during childhood. In fact, in most of the laboratory studies, cancers appeared late in life, regardless of whether exposure was begun in infancy or in early adulthood."[51]

C. Toxic Sensitization Is Increasing, Destroying Normal, Active Lives.

A medical problem that is receiving increasing attention and which deserves special consideration is the non-specific, debilitating syndrome of chemical sensitivity. Often (but perhaps not always) the result of some acute or traumatic exposure, victims suffer the triggering of symptoms and observed sensitivities at very low levels of chemical exposure. A recent report to the New Jersey State Department of Health by Dr. Claudia Miller at the University of Texas Health Science Center and Dr. Nicholas Ashford at the Massachusetts Institute of Technology contains the most comprehensive study of this topic to date, and concludes that "existing evidence does suggest that chemical sensitivity is increasing and could become a large problem with significant economic consequences related to the disablement of productive members of society".[52] For individual victims, the use of pesticides threatens their health daily and forces lifestyle alterations. Kevin Ryan, an extremely bright and articulate 11 year-old was invited to offer testimony at the Oversight Committee Hearing on Lawn Care Pesticides in March of this year. He spoke of his symptoms when exposed to lawn care pesticides in his neighborhood, "numbing and tingling of arms and legs, muscle and joint achiness, chest pressure, respiratory problems, nausea, severe stomach pain, diarrhea, brain symptoms, loss of memory, lack of concentration, irritability, depression and fatigue." He goes on to lament missing out on playing in his own yard with neighbors or playing baseball with friends. "During the months of March-June and August-October, my mother, brother, and I must vacate Illinois and go west, to the high elevations in Colorado where the air is clean. I have to leave my home, my friends, my school, and my father (since his job is in Chicago) just because the people in my town don't want any stupid weeds in their stupid lawns, and the government allows them to purchase these chemicals."

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[48] Gold, E. et al. (1979). "Risk Factors for Brain Tumors in Children." *American Journal of Epidemiology* 109(3):309-319.

[49] Lowengart, R. et al. (1987). "Childhood Leukemia and Parents' Occupational and Home Exposures." *Journal of the National Cancer Institute* 79: 39.

[50] Reeves, J.D. (1982). "Household Insecticide-Associated Blood Dyscrasias in Children." (letter) *American Journal of Pediatric Hematology/Oncology* 4:438-439.

[51] NRDC.

[52] Ashford, N.A. and C.S. Miller. (1989). *Chemical Sensitivity: A Report to the New Jersey Department of Health.*

IV. Farmworker Children Suffer Disproportionately High Risk

Perhaps the greatest pesticide exposures experienced by children occur on the farm. In addition to working in the fields themselves, farmworker children can be exposed through prenatal maternal exposure, from being in the fields where their parents work, contact with pesticide residues on parents' clothing, and living in migrant camps next to fields being treated. While industrial child labor was outlawed in 1938, only a few states have set a minimum age for child farm labor outside of school hours and little is done to enforce these laws.[53] Child labor is important in agriculture. A report by the American Friends Service Committee (1970) found that one-fourth of all farm labor in the U.S. is performed by children.[54] In 1981, according to the U.S. Dept. of Labor, an estimated 397,000 children, aged 8 through 15, worked in agriculture as compared with 1.2 million adults.[55]

Labor-intensive crops are also those that receive heavy pesticide application. Of the one billion pounds of pesticides used annually in agriculture in the U.S., 800 million pounds are applied to approximately 20% of the total crop acreage; most of these crops involve use of field labor on a seasonal basis. Furthermore, over 50% of farmworkers are hired for harvesting

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[53] Fuentes, J.A. (1974). "The Need for Effective and Comprehensive Planning for Migrant Workers." *American Journal of Public Health 64(1):2-4.*

[54] American Friends Service Committee. (1970). *Child Labor in Agriculture.* Report done in cooperation with the National Committee on the Education of Migrant Children. Summer, 1970.

[55] DiPerna, P. (1981). "The Lethal Cloud of Indifference." *The Nation.* June 27, 1981.

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operations, which involve contact with foliage during periods of high pesticide application.[96] EPA and the Occupational Safety and Health Administration (OSHA) has set some standards for worker safety, but these are based on adult exposure only.

V. Regulatory Policy Has Failed To Address The Reality Of Children's Sensitivity To Toxic Exposure.

Though the previous testimony highlights the scientific communities' understanding of the behavioral and physiological basis of children's susceptibility, the regulatory apparatus has failed to act on this.

A. Information Necessary To Accurately Assess Children's Nondietary Exposure To Pesticides Is Not Available to the Decision Makers.

A January, 1988 EPA internal memorandum on lawn pesticides acknowledges that the information necessary to assess public exposure to lawn care pesticides is lacking.[97] An August, 1990 EPA internal memorandum from the Non-dietary Exposure Branch expresses concern that their assignment for the current reregistration effort involves only 13 summaries which represent only worker reentry studies. The author questions, "Am I to assume that the current definition of "reregistration" excludes any further evaluation of data for the occupational areas addressing mixers, loaders and applicators? Am I further to assume all residential exposure, including all indoor and lawn pesticide uses, are likewise excluded until reregistration is completed in 9 years? "[98]

B. In The Dietary Realm, Data Required To Assess Health Risks in Children May Be Available But May Not Be Used in Regulatory Decision Making.

Utilizing the capabilities of the Tolerance Assessment System (TAS), EPA can generate dietary health risk assessments for 22 population subgroups, including infants and children. In a review of the tolerance setting process last year, U.S. GAO found that EPA usually develops separate cancer risk estimates only for the overall U.S. population, even though population subgroup's exposure to pesticide residues may be higher.[39] The GAO investigation describes an internal EPA memo, dated March 1986, which recommends that among the subgroups relevant to toxicity data, the subgroups with the highest exposure should be used as the basis for regulatory decisions. In most cases, according the document, the subgroups selected for decisions would be infants and children, except for decisions concerning birth defect and reproductive effects which may be relevant only to certain subgroups. However, EPA has failed to adopt this as policy. According to Office of Pesticide Program (OPP) officials, OPP prefers to gain experience with individual cases before setting overall policy. EPA now has four years of experience in using Tolerance Assessment System (TAS) subgroups data. TAS has been used to assess about 185 pesticides. At publication of the GAO report last year, separate cancer risk estimates for age subgroups, such as infants and children, had been considered in regulatory decisions for only three carcinogenic pesticides. For these pesticides, EPA had cancer data, other than that routinely required, which indicated young animals developed tumors.

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[96] Wilk, V. (1986). *The Occupational Health of Migrant and Seasonal Farmworkers in the United States*. Farmworker Justice Fund, Inc. Washington, DC.

[97] U.S.-EPA. *Lawn Pesticide Policy Group Briefing Paper*. Internal Document. January, 1988.

[98] U.S.-EPA. Office of Pesticide and Toxic Substances. Memorandum re: Additional Worker And Residential Exposure Support For FIFRA '88. August, 1990.

C. Negligible Risk Policy Harms Children.

Overall, we recognize the need for a holistic and thus more realistic approach to hazard assessment which considers the risks of pesticide exposure encountered through all possible routes, including diet, inhalation, and across the skin. Unfortunately, the data necessary to make such an assessment are rarely available and are not required by the regulatory system. In fact, recent proposals to address residues of carcinogenic pesticides in processed food seek to replace statutorily the pesticide portion of the Delaney Clause or "no additional cancer risk" provision of the Food, Drug and Cosmetic Act (Section 409) with a "negligible risk" standard. President Bush has proposed such a change in his Food Safety Plan, announced in October, 1989 and a numbers of bills are pending before Congress. EPA has actually adopted a regulatory interpretation of the Delaney Clause which establishes a negligible risk standard tied to an "acceptable" incidence of cancer. This interpretive rule, published in October, 1988, now being challenged in court, will further undermine protection of children specifically and the general population.[40]

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[39] U.S. General Accounting Office (GAO). *Guidelines Needed for EPA's Tolerance Assessment of Pesticide Residues in Food*. Statement Before the Subcommittee on Health and the Environment, Committee on Energy and Commerce, House of Representatives. May 17, 1989.

[40] 53 FR 411050, October 19, 1988.

This standard relies on uncertain risk estimates which seek to calculate the additional cancer risk posed from a single pesticide, ignoring real world risks, where multiple pesticides are encountered. Exposure via all routes, dietary and non-dietary, are not addressed.

Attempts at managing risks under a "negligible risk" policy ignore the fact that the last three decades have confirmed the scientific basis of the Delaney Clause and our inability to quantitatively define carcinogenic risk. Cancer mechanisms are not completely understood, but all scientifically acceptable theories preclude measuring or predicting a "safe" level of exposure to any carcinogen below which no individual or population group will develop cancer. Recognition of this forms the basis of the Delaney Clause standard of no additional cancer or "no induction of cancer."

Cancer is a killing and disabling disease of epidemic proportions. Cancer now strikes one in three persons and kills one in four.[61] According to the Centers for Disease Control (CDC), the chances of an American child born in 1985 developing cancer is one in three, with 20 percent experiencing premature death. According to CDC, cancer remains the number two cause of premature deaths, ranking only behind accidental injuries.[62]

D. Inadequate Neurotoxicity Testing Requirements Represent a Serious Deficiency.

Since 1986, serious efforts to improve neurotoxicity testing requirements have been attempted. In 1987, The Center for Science in the Public Interest (CSPI) and 11 other groups and individuals petitioned EPA to develop methods for assessing neurotoxic effects of active and inert ingredients in pesticides.[63] In May 1988, the Senate Agriculture Committee's report accompanying the FIFRA amendments contained a section that would have required the EPA Administrator to "develop methods for testing to accurately detect neurotoxic and behavioral effects of pesticides and their ingredients," and "as such methods are developed, require to the extent appropriate and necessary that data from such testing be submitted by persons seeking to obtain or maintain pesticide registrations." This provision was not included in the amendments finally enacted, but the House Agriculture Committee's report that became law noted the deficiencies of EPA's current neurotoxicity testing and called for improvements.[64]

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[61] Epstein, S.S., "Losing the War Against Cancer: Who's To Blame and What To Do About It," *International Journal of Health Services* 20(1):53, 1990.

[62] *Philadelphia Inquirer*, "Study: 1 of 3 born in '85 to get cancer," July 18, 1986.

[63] U.S. Congress, Office of Technology Assessment.

VI. Conclusion and Recommendations

Only By Protecting The Most Vulnerable, Can The Public's Health Be Protected with Assurance.

(i) A policy that protects only a portion of the public's health is not a true public health policy. Infants and children, who are especially vulnerable to toxic insults, have no voice in public policy and are unable to control their toxic exposures. EPA should be required to immediately establish a policy that tolerance decisions are to be based on the most highly exposed subgroup, which in many cases will be infants and children.

(ii) Information regarding the differing risks among population subgroups must be provided to the public in the rule-making procedure. Much of the current public mistrust of government regulations comes from revelations that information has been withheld.

(iii) The pesticide portion of the Delaney Clause (Section 409 of the Food, Drug and Cosmetic Act) must be preserved and expanded to ensure that children and the general populations are not exposed to cancer causing pesticides.

(iv) Neurotoxicity testing requirements must be revised. The nature of long-term neurobehavioral and psychological effects of exposure to neurotoxic pesticides is unresolved and requires further investigation. In their report titled *Neurotoxicity: Identifying and Controlling Poisons Of The Nervous System*, the Office of Technology Assessment writes, "limiting the use of neurotoxic pesticides would be a straightforward way to control exposure."[65]

(v) Eliminate the current risk-benefit standard in FIFRA and replace it with a health-based standard to ensure that children do not face the unnecessary risks that they currently do.

Issues of pesticides and children raise critical questions about the adequacy of the statutory and regulatory system governing the use of toxins and the necessity of using toxic materials for particular pest management needs.

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[64] Ibid.

[65] Ibid.

However, as public attention is drawn to the widespread and unnecessary public exposure to pesticides, industry is engaged in an effort to challenge the science that underlies the urgent need for governmental action to restrict pesticides. For example, in recent months we have heard charges that laboratory animals which are used to determine the ability of materials to cause cancer in humans are not an accurate measure. Those in industry or industry-supported organizations who claim that laboratory animal studies – which are endorsed by all independent scientific institutions including the International Agency on Research on Cancer (IARC)– cannot be used to define human health hazards are attempting to divert attention from a national health crisis.[44] The laboratory studies have in fact proved to be a useful indicator of effects in the human population.

Our task as a nation is to move ahead with protective environmental health policies that give children the strength and health to realize their dreams. To neglect children's health is to neglect the country's future.

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[44] IARC reviewed 44 chemicals known to cause cancer in humans and found that 84 percent of those chemicals were also found to cause cancer in laboratory animal studies. The other 16 percent of the chemicals had incomplete lab testing. Wilbourn, J. et al., "Response of experimental animals to human carcinogens: an analysis based upon the IARC Monographs programme," *Carcinogenesis* 7(11):1853-1863 (1986).

Chairman MILLER. I believe that the statement was made that nobody has ever died from pesticides. Do you agree with that?

Mr. FELDMAN. I will take that one first. No, that is simply not true. There are a number of cases of farm worker deaths. A member of our board of directors, the wife of a husband who died on a golf course from exposure to chlorpyrifos, which is one of the best documented cases of pesticide death in the country, only because he was a lieutenant Navy pilot and they brought the efficiency and the equipment of the Navy to bear on that investigation.

The point is, however, and implicit in what these people are saying is that it is not useful to use laboratory animal studies to deduce or extrapolate adverse human effects, which is simply not the case. I cite in my testimony a study by an international agency——

Chairman MILLER. I understand that.

Mr. FELDMAN. There are deaths. More importantly, because we don't have a pesticide incident monitoring system in this country, which you have to remember, we are not tracking either the disease rates or the death rates, there is no repository. We have to rely on animal studies to make the extrapolations.

Chairman MILLER. Dr. Pollack.

Dr. POLLACK. I don't believe that no one dies either. For one thing, in Central America, people from here who have worked with pesticide applicators there and farm workers know that many people do die. Also, we are talking about immediate deaths, but I think the farmers in our part of the world are getting to be very concerned when a few of them die of cancer about whether that may be linked. What we really don't know much about is, what are the long-term risks contributing to deaths?

Chairman MILLER. Dr. Wilkinson.

Dr. WILKINSON. Mr. Chairman, the figures at the moment indicate that somewhere on the order of 30 to 50 people die each year from pesticide poisonings. Most of these are, in fact, accidental deaths or due to gross misuse, suicides, this kind of thing. On the other hand, we have very few records available in this country that indicate how many people are in fact being injured on a chronic basis.

I would agree that, with respect to workers in particular, occupational exposures, there is no question in my mind that there are people at risk. If we are talking about the general population, I don't believe there is anyone who has ever died from pesticide exposure as a result of exposure as a member of the general population.

Chairman MILLER. Probably six or eight million golfers would disagree with that point of view.

The fact is, you have all testified we really don't know, because, in my home state, where I have seen workers sprayed during the process of work, and it happens in other areas of the country, we don't know the impact of that, because no one is watching out after them, no one is recording it, no one is monitoring it. So we don't know. I guess you are defining workers and general public differently, except that workers encompass millions of individuals in this country.

Dr. **Wilkinson**. Mr. Chairman, may I just add that we don't know for sure. There is a great deal of uncertainty involved in this whole risk assessment process. The fact is that although we don't know, why do we continue to assume that in fact these people are dying? In other words, there seems to be an implicit trust out there that if only we did the right tests we would find the reasons why these people were dying. There is absolutely no evidence. There is absolutely no evidence that pesticides are involved in deaths or increases in cancer or other adverse health effects on the U.S. population.

Mr. **Feldman**. With all due respect——

Chairman **Miller**. Yes, with all due respect from this side, you went from 30 workers to people in the general population, now to the entire U.S. population, there is no evidence.

Dr. **Wilkinson**. There is no evidence.

Chairman **Miller**. So you are back to the notion that nobody has ever died from pesticides.

Dr. **Wilkinson**. Yes. But I am back——

Chairman **Miller**. Yes?

Dr. **Wilkinson**. I believe that nobody has ever died from pesticides in the general population; correct. At least I have never seen anything documented. I don't know about it. But you can't prove that nobody ever died from pesticides; I agree.

Chairman **Miller**. For years, we couldn't prove that anybody died from exposure to asbestos because the people that had the figures weren't telling.

Dr. **Wilkinson**. But Mr. Chairman, why do——

Chairman **Miller**. They weren't releasing the health studies for years on their workers.

Dr. **Wilkinson**. Why don't we look at the good side of life and think that we are living longer than ever before? Our health and well-being is better than at any other time in our history, and yet we worry about all of these things that might be happening, when in fact there is no evidence that they are happening. It is the other way around, in fact.

Chairman **Miller**. Well, except that this committee has received testimony and other committees have received testimony. I have done it on the Labor Committee, where organizations, professional organizations, scientific organizations, have closely monitored, watched, and worked with select populations. In fact we do find a relationship between the exposures and tumors.

We listened to a young woman the other day, rather her parent, from central California, regarding the onset of tumors. Now, I don't know whether she will be recorded or not, and I don't know for a fact that it is related to pesticides, except we find that is one thing that has invaded her life on a consistent basis as the child of a farm worker.

So the suggestion is, because her parents were able to bring us a bounty of tomatoes, we should be joyous forever? The fact is, we ought to find out whether or not her parents are bringing tomatoes by subsidizing them with their broken backs and bad health.

Dr. **Wilkinson**. Of course, I don't disagree.

Chairman **Miller**. The sun came up today, but the world isn't all well, though I'm damn delighted the sun came up. And I think there is an obligation, especially when we see large segments of the working population—however you want to dissect this population—that are exposed in violation of laws which the Congress has set as a matter of public policy.

We have a right to know whether now that we have made that public policy decision, we are now exposing people to harm in violation of that law. As we continue to receive documentation in this committee, that is not an insignificant number of people.

Dr. **Wilkinson**. Mr. Chairman, I am not trying to say there are not some instances that can be resolved and improved; obviously, there are. I am just saying that we always tend to take this doomsday view of the world.

Chairman **Miller**. No, no. It is not a doomsday view of the world; it is legitimate inquiry about the protection of citizens from what may be unnecessary exposure to harm. I appreciate this argument. We all understand there is radiation in the environment. There is background radiation, but we try to minimize unnecessary exposures. We all understand that there are carcinogens that naturally exist in fruit, in fiber, and what have you, but we try to minimize, if we can, unnecessary exposures, if we believe that those agents can be carcinogenic.

It is not doomsday. That is different. If I were to read your testimony on its face, it would say, don't have any inquiry because you don't like the nature of the inquiry.

Dr. **Wilkinson**. No, I didn't say that.

Chairman **Miller**. That is usually what has slowed down most of the inquiry at the governmental level. It is not a doomsday theory; it has nothing to do with gloom and doom. It has to do with, as we just heard, three million children exposed. It has to do with tens of thousands of children who migrate across this country being exposed. That is a legitimate question of public policy and our obligation to those children.

Dr. **Wilkinson**. Mr. Chairman, we heard this morning several very important issues that we should approach and address and, if possible, resolve; we have to. The lead issue is one. We heard of the tobacco smoke issue. There are other issues that have to be approached and identified and resolved.

Chairman **Miller**. You asked, why the inquiry? You asked at one point, what's going on? There is no evidence, not a whit of evidence. You know, representatives of the tobacco industry sat on the other side of witness tables all over this town and today still suggest there is no link. They take out national advertisements. "There is no link." That is an interesting line from the industry. It has no credibility.

Dr. **Wilkinson**. Absolutely. And there are links, obviously. There are established links, but not with some of the issues we are talking about today.

Chairman **Miller**. They came about as a result of those kinds of inquiries.

Mr. Sikorski.

Mr. **Sikorski**. Thank you.

When I came here eight years, the tobacco people came into my office and said, very similar to what you said, "There is no evidence that any health effects have been proven." They said no epidemiological studies, no—what is the other word that you used?

Dr. **Wilkinson**. Scientific evidence.

Mr. **Sikorski**. No scientific evidence whatsoever, and made the absolute statement. You made the absolute statement that there is no evidence of any deaths or danger to people from pesticides, to children from pesticides.

Dr. **Wilkinson**. I said from ingesting.

Mr. **Sikorski**. On food.

Dr. **Wilkinson**. Food and water contaminated with the residues that exist at the present time.

Mr. **Sikorski**. The National Academy of Sciences says there are a million people, a million Americans are going to die because of pesticides ingested through food. Those scientists, you know, didn't dream it up. And we know, do we not, that when you say pesticides, we are talking about known mutagens, carcinogens, teratogens, neurotoxins that have been proven to be these things in laboratory experiments on animals; right?

Dr. **Wilkinson**. Correct, at high levels, in many cases.

Mr. **Sikorski**. There are ways that scientists have to do these tests. What you are hiding behind—and I appreciate your "don't worry, be happy" because there is an element of not being paralyzed by the perils of daily life. It is important that we voice a balanced concern, we put this into perspective. There are malnutrition problems; there are physical and sexual abuse problems regarding children.

This committee has gone on the spectrum of issues, but that does not discharge the responsibility of public policymakers, scientists, and others to focus attention on this and doesn't—and you will appreciate this, I know; I've talked with you—does not diminish the sorrow that parents have when a child dies of cancer that at least may be caused by chemical toxins in the environment.

Chairman **Miller**. Yes, Dr. Pollack.

Dr. **Pollack**. Actually, I appreciate your having this hearing because I think the issue isn't just about death, and it isn't really even just about children. I think they have frequently, certainly in the history of occupational medicine, served as a vehicle for improving the health of everybody. The issue here isn't just death and it isn't just farm worker children.

When I worked for a regulatory agency before going to medical school and lived in this city, my exterminator for my building came one day and gleefully informed me that the reason he had been so successful in eradicating the roaches was that they were using things that were twice as strong as what was allowed. I don't think that is an uncommon situation.

You are not just talking about deaths; you are talking about asthma attacks; you are talking about a whole variety of health effects. Also, I think there is a real potential for something positive to happen and that perhaps we shouldn't get mired in just why you shouldn't do this.

There are farmers concerned about pesticide use who would like to have information about how else they might conduct their agriculture. I know that is true in New York State, but the extension agents don't know very much about other methods. There are things that could be done about that.

The farmers don't know where to put their pesticide containers when they are done, and so they get left around where children play in them, in the pools where they mix pesticides. If they had a place to send those back to the manufacturers, which one company does, that would be something positive. If the applicators, who are supposed to be trained and certified, were really applying them in the way that they are supposed to, then the whole public would be at less risk.

I think there are things that are deserving of being addressed and looked into.

Mr. **Sikorski**. Mr. Chairman.

Chairman **Miller**. Yes.

Mr. **Sikorski**. I want to commend all of you for participating in this and assisting the select committee. I do want to focus on this: Dr. Wilkinson, you have used, as kind of—the opposition is blanket stating, making blanket conclusions, you said, and they are exaggerating their concerns. But then in your statements you use blanket statements, and you weave throughout this in a very—do you do expert witness? Are you an expert used by people in—

Dr. **Wilkinson**. In legal litigation support work, you mean, on this sort of stuff?

Mr. **Sikorski**. Yes.

Dr. **Wilkinson**. Sometimes.

Mr. **Sikorski**. You testify for whom, the defense?

Dr. **Wilkinson**. I have testified for both plaintiffs and defense. I have worked for plaintiffs, as well.

Mr. **Sikorski**. Because what I see in this, as a former litigator, is very nice, positive testimony, couched with—you move back and forth on the issue. "Conventional wisdom has developed that children are always more sensitive than adults to the action of pesticides and other synthetic chemicals—highly emotional—the issue that has both angered and frightened people." You set up the straw child of "always more sensitive than adults."

I have not heard anyone, even the biggest advocate of harsh, strict activity on the part of the federal government or any government to regulate pesticides, ever say that children are always more sensitive than adults to the action of pesticides and other chemicals.

Then you go on to say, you know, "It is just not true. There are some situations in which infants might be at a greater risk to a given toxin." Then you go on, "There is nothing to suggest that any problem exists—don't support that." You say, "There are no data to suggest that any problem exists—don't support that." You say that "Data on child sensitivity is extremely difficult to obtain," which is probably the most honest conclusion that any side can say on this is that we don't have the data.

You are making blanket statements all the time. You say that "Advancement of scientific tests now allow measurements per million, per trillion, even quadrillion. This may unduly heighten concern." I can agree that it might, but what is happening now is that the EPA and the FDA and the USDA are not testing using these modern treatments. We are letting pesticides and chemicals go throughout the system without testing them.

You say, on the bottom of page two, that "The worst-case scenarios are used exaggerating real risks to kids." The regulatory system has used the worst-case scenarios to support continued use. You seem to disagree with the idea that we should look at consumption patterns, especially kids' consumption patterns. Apparently, the regulators don't do that, except in some instances when it cuts for the pesticides to continue in the marketplace or to continue unreviewed.

The regulators use the application as recommended by the manufacturers. Dr. Pollack just mentioned, and we all know, that we have kind of a mentality: if a little is good, that more is better, and most is great. There are a lot of people—we had in front of the health committee back in the sulfites on the food, we had a lot of food service workers who thought if you put a tablespoon in two gallons of water, which was called for, three tablespoons was better, and people died from eating stuff at snack bars, salad bars.

You also said, "Kids consume only two to three times more of most foods than adults consume." How do you know that?

Dr. WILKINSON. May I respond to some of the things.

Mr. SIKORSKI. Respond to that question and then go back and treat all of them.

Dr. WILKINSON. You can actually come up with estimates on that based on what we do know about what kids eat, USDA diet figures, and so on and so forth.

Mr. SIKORSKI. The fact is that you knock down again—let me see if I can find exactly how you say it—but you make a blanket statement that it is just not true. You make a blanket statement in counter to—you criticize your opponents as stressing the very worst, and then you say kids consume only two to three times more food than adults. There isn't any evidence supporting that statement; it's your opinion.

Dr. WILKINSON. Mr. Congressman, in a hearing of this type, you must agree we are here discussing a very complicated subject in a very short period of time.

Mr. SIKORSKI. No, but you criticize other people and then you turn around in the same breath, in your oral testimony, and do the same thing. There isn't any support for your statement. Do you agree that infants in this country eat 16 times as much applesauce as adults?

Dr. WILKINSON. Sixteen times as much?

Mr. SIKORSKI. Yes.

Dr. WILKINSON. Some probably do.

Mr. SIKORSKI. Infants, on average.

Dr. WILKINSON. Some may. Again, you can't generalize.

Mr. SIKORSKI. But do they only do two to three times as much?

Dr. WILKINSON. You can't generalize.

Mr. SIKORSKI. You just did. That's my complaint. You complain other people are generalizing, and you complain other people are making exaggerations, you complain other people are making blanket statements, and your testimony is full of exaggerations, blanket statements, and generalizations.

Dr. **Wilkinson**. I apologize. If we could take a whole day to discuss this, we could go into a lot more detail. It is very difficult to cover a complex area like this in a few minutes.

Mr. **Sikorski**. I appreciate that, and I appreciate your position. I think it is helpful to this discussion. But, once again, you make these charges of the other people, do it yourself, and your conclusion, though, is that we shouldn't take any action.

Dr. **Wilkinson**. May I ask a question, Mr. Congressman? The other side, if you want, are making these kinds of claims all the time, terrible claims that I have heard this morning.

Mr. **Sikorski**. You make them all the time, too.

Dr. **Wilkinson**. But why should they not be responsible for backing up some of their claims with data when you are asking me to do that?

Mr. **Sikorski**. I think they are, and they have to, as you do when you are.

Dr. **Wilkinson**. But it never happens.

Mr. **Sikorski**. It didn't happen in your case.

Dr. **Wilkinson**. I could do that.

Mr. **Sikorski**. And you know why. You have made mention, because there is very little data on this stuff, very little research, kids have been ignored. That is why we are having this hearing in the first place. Beyond that, you know that we are not talking about people dropping over dead in produce fields, golf courses; we are talking about chronic, long-term exposure and cancers and mutations that show up generations later, cancers that show up decades later.

It is a great shield to protect those responsible, if there is responsibility, because it is very difficult to prove cause over decades. I have had this fight before. We have a community right-to-know provision. We now have an air toxins provision that will probably be made law. Five years ago, when I did the community right-to-know fight on the floor, and we only won by one vote, all the people came out and used the same arguments that you are using: "We don't really know. There isn't any evidence. It is very difficult to find linkage."

We made them report. We said, the heck with linkage, just report how much you expose each year. Then we find out there are over two billion pounds of chemical toxins that are carcinogens, mutagens, teratogens, and the rest, that are being poured into the air in America every year by chemical companies. That is six times what they told us was the absolute height of what was being discharged by them.

Dr. **Wilkinson**. May I make one plea, I guess in my defense, all I am basically asking is that when we are addressing these issues that are many and complex, we have to set some kinds of priorities. We don't have unlimited resources in this country to identify and resolve all of these issues at once. We have to set priorities.

All I am asking is that in setting priorities that we are very careful not to squander these precious resources and that we address issues that, based on sound science, actually are an established problem of some kind. We can't squander these things away, this money away, these resources that really aren't a problem until they can be identified as a problem.

Mr. **Sikorski**. Tell me how you identify three decades later problems.

Dr. **Wilkinson**. It is a real problem. I am not belittling it, but I am just saying we have to be extremely careful.

Chairman **Miller**. That was the fight over reregistration.

Dr. **Wilkinson**. Yes.

Chairman **Miller**. So the notion is you just go ahead and you put them on the market. You haven't looked at them with the impact on pregnancy. You haven't looked at the impact on children. You haven't made these decisions. And the argument can be made, well, there are just not enough resources to do this, so maybe we can do 1 out of 10 chemicals, or 1 out of 10 additives, or 1 out of 10 that come to the market.

I think, as Mr. Sikorski pointed out, the intolerance of that is growing in the American public; again, when there is also a study on the other side that in some instances, demonstrate that, the dosages aren't terribly beneficial to the farmer or to production, where the combination of dosages may be destructive to the environment beyond humans.

Dr. **Wilkinson**. But please don't throw science out the window in your deliberations, that is all I am asking.

Chairman **Miller**. I want to know, who would suggest——

Mr. **Sikorski**. We are saying, use science. Where is the science that supports the use of chemicals?

Chairman **Miller**. Who suggested throwing science out? You come here with a bundle full of arguments that are not even relevant to the discussion.

Dr. **Wilkinson**. Well, I'm sorry.

Chairman **Miller**. There is nobody on this committee who suggested we throw science out. We have not yet had a single witness who has suggested we throw science out. We have not had a single witness who has not provided documentation for their statements so far, except for you. That is not the purpose of the inquiry of this committee and it never has been. It is an interesting argument to set up that somebody out there wants to throw science out, but that is not the purpose of this inquiry.

Mr. **Sikorski**. What we are saying is, don't the chemical companies have a responsibility to scientifically prove that what they are marketing is safe?

Dr. **Wilkinson**. You can never prove that anything is safe. That's the problem.

Mr. **Sikorski**. Don't accuse us of trying to throw science out. We are saying, science should be used by those who are marketing products that are making the argument, don't throw science out the window. They should use science, updated science, to prove or to disclose limited risk of the chemicals that they are peddling in the marketplace.

Dr. **Wilkinson**. I agree. To the extent that science has its limits, they do that.

Mr. **Sikorski**. Tell me how those grandfathered pesticides are being proven by the chemical companies that are peddling them in millions of tons around the world, tell me how they have borne the burden of science.

Dr. Wilkinson. First of all, science is an evolving thing. What we accept now, the criteria we accept for acceptance of data is quite different from what it was last year or five years ago.

Mr. Sikorski. And they are not carrying the burden of using that science. We are saying, use the science. How are they carrying that burden of using the science?

Dr. Wilkinson. It is an ongoing process.

Mr. Sikorski. They have thrown science out the window.

Dr. Wilkinson. No, they are not. They are trying to hit a moving target all the time, and this is the basic problem. I am not saying that the chemical companies, in many cases, have got a lot to be proud of there.

Chairman Miller. Unfortunately, they have done it with crop dusters. That is who they are hitting. You know, when they went to the reregistration, they came in and were looking for the broadest possible exemption from dealing with modern, updated science, and we were supposed to continue those products on the market.

Dr. Wilkinson. In many cases, Mr. Chairman, those products will not be on the market anymore, because the chemical industry cannot afford to do a lot of the tests that are required to bring them up to date. I think, sooner or later, I have a feeling that agriculture in this country might ultimately suffer. It's not in the next few years, for sure, but it could have some serious implications in terms of the number of materials that are actually available at the present time.

Mr. Sikorski. Mr. Chairman.

Now, once again, science should not be used because it is too expensive to the chemical companies. Is it science out the window, or is it science in the window?

Dr. Wilkinson. I didn't really say that, at least I hope I didn't.

Mr. Sikorski. Yes, you did.

Dr. Wilkinson. What I was saying was that in terms of the benefits, economic benefits, that the industry can gain, it is simply not cost-benefit worth it to go ahead and do a lot of these tests that are required for reregistration.

Mr. Sikorski. What about the advocates? They say it is not cost-beneficial to kill people using these chemicals, or maim them, or whatever, so, therefore, they should not have to carry the burden of the scientific evidence. You find that a terrible argument. You call it throwing science out the window. But when the chemical companies use the economic rationalization to throw science out the window, you are not appalled by that.

Dr. Wilkinson. I am appalled at anybody trying to throw science out the window in issues of this type.

Mr. Sikorski. Thank you.

Chairman Miller. Dr. Pollack, if I can ask you, since your oral testimony was somewhat different from your written testimony, if you could just follow up with this, because the migrant area is one of the areas we plan to pursue somewhat more in depth a little later. So if you can fill in your oral testimony, I would appreciate that very much.

Dr. Pollack. We will, yes. Thank you.

Chairman Miller. Congressman Holloway.

Mr. **Holloway**. Dr. Pollack, I am sorry that I missed the testimony, but I had read your speech or your testimony beforehand. On page four of your testimony, you make some vague and rather rambling assertions about the conditions of migrant workers. Your testimony is incomplete and leaves a lot of inaccurate impressions. You may want to try to answer my question a little bit as I go along here.

We have a lot of laws on the books today which cover this. I mean, basically, I am in this type field of work. I don't use migrant workers, and I never intend to use migrant workers, but I think, for the record, with OSHA in place and FIFRA, and many of the other laws that we have passed, I guess I would like to hear—and I'm sorry that I was not here to hear the questions from the other congressmen, but it just amazes me how people can come here and try to make the laws of the land look as if we are not doing any of the things that we need to be doing.

I visited some migrant workers from Jamaica in the sugar industry just this past January. I went back and watched a terrible segment on "60 Minutes" that basically totally was opposite of what I had looked at. For the people who seem to want to beat up on this, I am not going to tell you that we don't have problems with our own people in our own country. But when do we end trying to put the impression that we don't have anything that protects people?

I believe that we have made tremendous leaps and gains, and probably these people are so much safer and so much better treated, and I think we even have laws that prohibit kids from even following their parents into the field, once they cross the line going to the field.

Dr. **Pollack**. No.

Mr. **Holloway**. Yes, there are.

Dr. **Pollack**. They are not enforced.

Mr. **Holloway**. Well, I'm not going to tell you they are not enforced. I don't think we have enough problems with them that if we are not enforcing them, I think there is a reason for it, that there are not that many cases that it is being done.

Dr. **Pollack**. May I just say, I am sorry you missed my testimony, though, because this was written a week ago to try to make your early deadline, and my testimony was actually somewhat different. I think that you would have been happier with that, because I made the point that there were many laws that existed already. I did make that point. Actually, OSHA does not cover farm workers, so that is unfortunately really not included.

I did make the point that the Fair Labor Standards Act, for one thing, although also there are exemptions for farm workers, was designed to protect that work in the fields, and specifically—well, children at work—and specifically prohibits hazardous occupations for children under 18. I think that is why Chairman Miller had just asked me to update my testimony because the written was somewhat different from the oral.

I do think it needs to be pointed out, the Jamaican situation is not at all the same. In New York State, we have Jamaican contract workers, too. The Jamaican government will not allow workers to come here unless at their camps there are functioning sanitary facilities. They have to have toilets. In eastern Virginia, on the Eastern Shore, they inspect once a year in June, and most of those people do not have working sanitary facilities. The Jamaican situation is different, because those are contract workers with the U.S. government, and that is not representative of the general migrant experience.

Mr. Holloway. All right. Let me tell you what happened to me personally. I am a nurseryman. I have never hired anyone other than American citizens till the unemployment department in one of my local towns called me and told me they had two identical twins they would like to find some work for. They were in a room, sleeping on a cot, and called and asked me could I help them, and either find them a job, knowing that I was a congressman, and I said, "Yes, if you will send them up, we will help them."

I worked these people two weeks. Within two weeks, the Labor Department was there to inspect their conditions, where they were living, where they were staying, and I was fined for two or three violations which I was not aware of. But to me, if within two weeks of having some migrant workers in my place I had people there to inspect, it tells me that we are doing something right in this nation. If we are not, well, I'm missing the boat myself.

Dr. Pollack. I guess the point of my testimony, which also was left out of here, is that, in New York State at least, the farmers are concerned, too. In the Hudson Valley, many of the farmers do their own spraying. It is not an issue just for migrant farm workers. The young farmers, especially, in that area and in the western part of the state, some of the older farmers have been the people who always did their own spraying. The issue of exposure to themselves and their families is something that I think the farmers have become much more concerned about.

One of the evidences of that is that, in western New York State, Farm Workers Legal Services provides education about pesticides to migrant farm workers. Last year the farmers called them up and said, "We are really concerned. Three of us have died of cancer in the last few months. We don't know if it is related, but we are worried. We would like you to come and educate us about pesticides."

I am not trying to turn it into a farmer versus farm worker issue. For many farmers in small farms, they do their own spraying, and it is just as much an issue for their own safety.

Mr. Holloway. Basically, you are saying the farmer himself should be regulated and told by the government how to operate his own family?

Dr. Pollack. No, I am not saying that. I think the farmers in New York State have a lot of questions and no one to answer them. One of the positive solutions I can see, which has come from my contacts with them, when they turn to agricultural extension agents to find out more about what alternative farming methods might be available to them to substitute for some of the pesticides which cost them a lot of money, there isn't anybody knowledgeable in the state. Cornell has, I think, one person who can talk to them, but there is no one who knows the answers.

Chairman Miller. Will the gentleman yield?

Mr. Holloway. Yes.

Chairman **Miller**. I think the point of the testimony, both written and oral, is, in spite of federal law, there are wholesale violations of that law. It is not Dr. Pollack who is documenting the wholesale violations of that law; it is almost every state labor department in the country. With respect to her contentions about the child labor laws and children working in garment factories and leather factories, and what have you, it is Elizabeth Dole who has documented the wholesale violations, called it a national scandal, had to put together a task force. The Reagan Administration had a task force because child labor laws were being violated in such a wholesale fashion.

In the State of California, under a Republican administration, they have had to go in and pull children out of the fields. They have to cite farmers for spraying children in the fields and workers in the fields. There has been gross violation of the laws and the protections. That does not mean that the laws need to be rewritten; the laws need to be enforced.

It is true all across this country. It is true in Delaware, and it is true in Texas. It is true in Virginia, and it is true in Maryland. It is true all across this country, in terms of where especially we see the use of migrant workers, be they foreign or domestic.

I spent a number of years as chairman of the Fair Labor Standards Committee, and we have enough reports to fill this room by regulatory agencies of every kind of governmental administration, Republican, Democratic, Conservative, Liberal, whatever the view, of the wholesale violations of these laws. In some instances, those violations also include the exposure of children and others to toxins.

Dr. **Pollack**. The point I was making was that the farmers have some concerns that currently are not being addressed and that we might do a better job of addressing. For instance, agricultural extension agents need to know more about alternate ways that farmers can spend less money on pesticides and find some other way.

The other issue that came up earlier is that I believe, as many people do, that most of the American public feels very strongly that if the government allows something to be sold it is safe. Therefore, it is very important not to gut the regulatory process because people really do believe that. You put people at even further, increased public health risk if you decide that you are not really going to regulate pesticides because it is too expensive for the manufacturers.

Mr. **Holloway**. Regaining my time, of course, from what Mr. Chairman said, I think we can take any issue we want and find any number of violations on anything in this country. I don't care if it deals with welfare and issuing it out, or if it deals with pesticides and spray. I think there is an illustration that can be drawn, in many, many cases, on any issue we bring up on this hill. So the fact that we have a hearing and I have a witness who comes and testifies something to me tells me there is a case, but it doesn't tell me that it is on a wholesale level out there.

Dr. **Pollack**. At the risk of boring other people because they heard this, and it isn't in here either, there were 112 people last year exposed in a situation similar to our 48 percent of our children, which is that they went back into fields where the manufacturers specify reentry times and farmers ideally know it is important to go along with that, but it goes along with their feeling that things are safe, that you don't really need to wait that long. There are not one or two; there are many people at risk.

This is a financial issue, too. Somebody paid those people's hospital bills.

Mr. HOLLOWAY. I don't know if the number you are talking about compares with how many that went into the fields. It might be 48 out of 452,000. I am not going to tell you that there are not those cases.

Moving on, I am going to read a little something here. Dr. Wilkinson said in his testimony, he said, "I am concerned that many of our regulatory priorities are currently being dictated by emotional and nonscientific claims and demands of a few highly vocal individuals and organizations," which I don't think there is nothing in this country that hits the media quicker and gets a bigger play than anything that deals with the environment today.

I think we strive for things, but I think this is a tremendous statement in your testimony. I want to say, having heard all the testimony from today's hearings, do you feel that our regulatory priorities should be set based on what has been said here today? I address Dr. Wilkinson in that.

Dr. WILKINSON. What we have heard this morning?

Mr. HOLLOWAY. Yes.

Dr. WILKINSON. I think some of the issues that have been raised this morning certainly should be considered by the regulatory agencies, but then I believe that the regulatory agencies should be considering all of the evidence available, the total weight of evidence that has to be balanced before decisions are made.

These are very complicated issues, and we can't just take one part of the data base and run with it. It is very easy to jump to conclusions, and often these are wrong conclusions. Therefore, we have to take evidence from here, evidence from there, pull it all together, carefully review it. And, sure, there are a lot of uncertainties. Life is a compromise in terms of making decisions, but we can come up with what is a best judgment decision based on the evidence available.

I think we are doing that rather well at the present time, with the exception of what I see as a trend in the last few years where I believe that media hype and emotion is starting to take over. It concerns me very much, as a scientist, that scientists are beginning to assume a seat further and further towards the back of the room in relation to making regulatory decisions. That is what concerns me.

Mr. HOLLOWAY. Just to follow up, do you feel that there is enough information forward on this that we should move forward here on the Hill, or do you feel that there should be much more information to come forward?

Dr. WILKINSON. Move forward in what way, Mr. Congressman?

Mr. HOLLOWAY. Well, I mean as far as if we are going to go to trying to make new regulations and pass new regulatory measures here that many we are going to be duplicating. I mean, basically, banning some of the pesticides that are out there that are not being properly used. I mean, the fact they are safe if they are properly used, but should we be banning them just because there are certain cases where they are not properly used?

Dr. **Wilkinson**. You missed some of the earlier discussion, and I am sure I come across to some members of the committee as being a very cavalier person who really doesn't have many concerns. That would be a long way from the truth. I really believe that if we have concerns and these can be identified and established, then we have to do something about it.

I don't believe that the evidence, in many cases, is there at the present time, at least with pesticides. I believe there is lots of evidence with some of the other issues you are considering. Lead is one example; obviously, that is a problem, and it is being resolved. I think with pesticides, don't let's just fire off and jump into this thing and start making all kinds of new laws and regulatory promises without having some facts to base those on. I don't believe that those facts are there at the present time.

Mr. **Holloway**. Thank you. I apologize for missing all your testimony. That's the way we operate here. It looks like we have to run and run into other hearings, but I have a lot of interest in this, particularly from my own, but—I am, as much as you, I think we have to be—certain things we do, but I think it doesn't mean that we are not soft-hearted to a point of realizing that there are problems or not looking at other ways of something we need to solve.

I think too often here we go out and pass regulations before we even have good information on what we are passing them on. I think that is the case of what we are trying to jump into here.

Thank you, Mr. Chairman.

Chairman **Miller**. Dr. Pollack.

Dr. **Pollack**. As a pediatrician, I just want to say, we were not here asking you to pass any new ones, but, for the health of children and adults, not to undo the regulatory process or weaken the regulatory process, and perhaps to strengthen what already exists under current law without passing new regulations.

Chairman **Miller**. Thank you. Thank you again for all of your testimony and the help you have been to the committee.

The committee will stand adjourned.

[Whereupon, at 1:23 p.m., the committee was adjourned.]

[Material submitted for inclusion in the record follows:]

Low-Level Lead Exposure and the IQ of Children

A Meta-analysis of Modern Studies

Herbert L. Needleman, MD, Constantine A. Gatsonis, PhD

We identified 24 modern studies of childhood exposures to lead in relation to IQ. From this population, 12 that employed multiple regression analysis with IQ as the dependent variable and lead as the main effect and that controlled for nonlead covariates were selected for a quantitative, integrated review or meta-analysis. The studies were grouped according to type of tissue analyzed for lead. There were 7 blood and 5 tooth lead studies. Within each group, we obtained joint *P* values by two different methods and average effect sizes as measured by the partial correlation coefficients. We also investigated the sensitivity of the results to any single study. The sample sizes ranged from 75 to 724. The sign of the regression coefficient for lead was negative in 11 of 12 studies. The negative partial *r*'s for lead ranged from −.27 to −.003. The power to find an effect was limited, below 0.6 in 7 of 12 studies. The joint *P* values for the blood lead studies were <.0001 for both methods of analysis (95% confidence interval for group partial *r*, −.15 ± .05), while for the tooth lead studies they were .0005 and .004, respectively (95% confidence interval for group partial *r*, −.08 ± .05). The hypothesis that lead impairs children's IQ at low dose is strongly supported by this quantitative review. The effect is robust to the impact of any single study.

(*JAMA*. 1990;263:673-678)

THE NEUROTOXIC properties of lead at high doses have been recognized for at least a century and are not a matter of dispute. In 1943, Byers and Lord[1] first suggested that childhood exposure to doses of lead insufficient to produce clinical encephalopathy was associated with deficits in psychological function. The question of low-level lead exposure has been studied widely over the past two decades and, in contrast to high-dose lead exposure, has been the source of considerable contention. Several methodological difficulties encountered in the conduct of these studies have contributed to the controversy. Among them are (1) selecting adequate markers of exposure or internal dose, (2) measuring outcome with instruments of adequate sensitivity, (3) identifying, measuring, and controlling for factors that might confound the lead effect, (4) recruiting and testing a sample large enough to provide adequate statistical power to detect a small effect, and (5) designing a study that avoids biases in sample selection.

A number of reviews of studies on the effects of low-level lead exposure on the neuropsychological function of children have been published.[2-6] The outcome of major focus in these reviews has been psychometric intelligence. The general approach was to provide narrative summaries in which the epidemiologic and statistical issues often received limited critical attention. Where quantitative synthesis was attempted, it consisted of a simple tally of those studies showing statistically significant effects (at the .05 level) vs those that did not. This approach gives undue emphasis to the individual study's *P* value and attaches equal weight to all studies without regard to their specific merits or flaws. The size of the effect measured in each study is generally ignored in the process.

The statistical techniques that have been subsumed recently under the rubric of meta-analysis offer a framework within which formal research synthesis can be conducted with more clearly defined methods and criteria.[7-9] In this approach, individual studies are treated as data points in a larger "meta-study." Summary measures from each study are pooled by one of a number of techniques, and quantitative inferences are drawn about the research questions of interest. The difficulties entailed in combining dissimilar studies ("apples and oranges") is a concern for any meta-analysis.[10] It points to the need for some measure of commonality in the studies that are being combined. At the same time, the usefulness and novelty of meta-analysis lies in the fact that it enables the investigator to combine the results of studies that *differ* in several respects, while addressing the same research questions.

The first meta-analysis of six lead-IQ studies was reported by Schwartz et al[11] in 1985. They used Fisher's aggregation technique to calculate a joint *P* value of .004 for the effect of lead on IQ in the six studies. Needleman and Bellinger[12] extended the analysis by Schwartz et al and also used Fisher's technique on pooled tooth and blood lead studies.

In the last few years, a substantial number of new epidemiologic studies from various nations, using more refined designs, larger sample sizes, and more sophisticated statistical techniques, have been reported. This presents an opportunity for a more comprehensive meta-analysis. Herein, 12 recent studies are reviewed and a quantitative synthesis of their results is presented. The major outcome of interest is full-scale IQ, although many studies also examined the effects of lead exposure on important functions such as school performance, reading ability, and classroom behavior. All studies reviewed employed multiple regression analysis in which the dependent variable (IQ) was treated as continuous. Lead exposure was classified by one of two methods: blood or tooth lead level. In contrast to earlier attempts, this analysis divides the studies by tissue analyzed and combines inferences within tissue groups. The question of possible bias in the obtained sample of studies (known as the "file drawer" problem) is addressed. Moreover, the aggregate effect of the exclusion criteria is assessed by performing an analysis that combines all of the initial 24 studies. The sensitivity of the results of this meta-analysis is further investigated by eliminating each of the included studies, one at a time, from the analysis and observing how this affects the conclusions. The statistical power of each study to find an effect is also computed.

This article presents a discussion of some methodological difficulties encountered in the studies reviewed, examines the critical question of effect assessment in pollutant studies, and concludes with comments on the difficulties entailed in drawing causal inferences from observational studies of lead exposure and intellectual development.

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Table 1.—Candidate Studies for Meta-analysis*

Study	Year	No. of Subjects	Tissue	Lead Level†	Data Analysis	Included/Reason for Exclusion	Comments	Lead Effect, P<.05
Kotok	1972	C=25; E=24	Blood	C=38; E=81	t test	No/G,D	A, B	No
Perino and Ernhart	1974	C=50; E=30	Blood	C<30; E=40-70	Multiple regression	No/H		Yes
Rummo et al	1979	C=45; E=45	Blood	C=23; E=61-88	ANOVA	No/G,D	A	Yes
de la Burde and Choate	1975	C=67; E=70	Blood	R=30-100	χ^2	No/D,E,G	C	Yes
Landrigan et al	1975	C=78; E=46	Blood	C<40; E=40-68	t test	No/D,E		Yes
McNeil et al	1975	C=37; E=101	Blood	C=28; E=58	t test	No/D	E, G, H	No
[illegible]	1976	80	Blood	PbB$\bar{X}$=33.2±9.1	Multiple regression	No/E	A, B	Yes
Kotok et al	1977	C=36; E=24	Blood	C=38; E=81	t test	No/G,D	A, B	No
Ratcliffe	1977	C=23; E=24	Blood	C=28; E=44	t test	No/E	A, B	No
Needleman et al	1979	C=100; E=58	Tooth	PbC=24; PbE=36 PbTC≤10; PbTE≥20	ANCOVA	No/H		Yes
Yule et al	1981	166	Blood	C≤13; E=13-32	Multiple regression	Yes		Yes
Winneke et al	1982	C=26; E=26	Tooth	PbTC$\bar{X}$=2.4; PbTE$\bar{X}$=9	t test	No/D	A	No
McBride et al	1982	106	Blood	C=2-8; E=18-30	ANOVA	No/D,E		No
Smith et al	1983	402	Tooth	PbT$\bar{X}$=5.1±2.8	ANCOVA	No/H		No
Winneke et al	1983	115	Tooth	PbT$\bar{X}$=6.2; PbB$\bar{X}$=14	Multiple regression	Yes		No
Harvey et al	1984	48	Blood	R=6.2-28.8	Multiple regression	No/F	A	No
Shapiro and Marecek	1984	193	Tooth	R=30-150	Multiple regression	No/E		Yes
Needleman et al	1985	218	Tooth	PbT$\bar{X}$=12.7	Multiple regression	Yes		Yes
Ernhart et al	1985	80	Blood	C=30; E=40-70	Multiple regression	Yes	A	No
Schroeder et al	1985	104	Blood	Median=30	Multiple regression	Yes		Yes
Hawk et al	1986	75	Blood	PbB$\bar{X}$=21; R=6-47	Multiple regression	Yes	A	Yes
Lansdown et al	1986	C=80; E=80	Blood	C=7-12; E=13-24	Multiple regression	Yes		No
Hatzakis et al	1987	509	Blood	PbB$\bar{X}$=23; R=7-63	Multiple regression	Yes		Yes
Pocock et al	1987	402	Tooth	PbT$\bar{X}$=5.1±2.8	Multiple regression	Yes		Yes
Fergusson et al	1987	724	Tooth	PbT$\bar{X}$=6.2±3.8	Multiple regression	Yes		No
Fulton et al	1987	501	Blood	GM=11.5; R=3-34	Multiple regression	Yes		Yes
Hansen et al	1987	156	Tooth	PbT$\bar{X}$=10.7; PbB$\bar{X}$=5	Multiple regression	Yes		Yes

*A indicates small sample; B, weak outcome measures; C, poor exposure measures; D, inadequate data analysis or reporting; E, inadequate or no covariate control; F, overcontrol; G, clinical levels of lead exposure (blood lead level >3.86 µmol/L); H, later reanalysis substituted (Needleman et al [1985] for Needleman et al [1979], Pocock et al [1987] for Smith et al [1983], and Ernhart et al [1985] for Perino and Ernhart [1974]); PbT$\bar{X}$, mean tooth lead value; PbB$\bar{X}$, mean blood lead value; R, range; PbTC, values for control group; PbTE, values for high-lead group; GM, geometric mean; ANOVA, analysis of variance; and ANCOVA, analysis of covariance.

†All tooth studies are measured in parts per million and all blood studies are measured in micrograms per deciliter.

METHODS

Data Collection

All studies on lead exposure and children's neurobehavioral development that were published since 1972 were examined for eligibility. The sources of candidate studies were a computerized MEDLINE subject search and a search of programs of meetings on metals, neurotoxicology, lead, pediatrics, and public health. Dissertation abstracts were also searched. Table 1 lists the studies identified in the search and presents summary data.

Studies were excluded for the following reasons: (1) Inadequate control of covariates reflecting socioeconomic and familial factors. (2) Overcontrol of factors that reflect exposure to the independent variable, lead. One study controlled for pica and peeling paint. (3) Inclusion of subjects with defined clinical lead poisoning (ie, blood lead levels >3.9 µmol/L). (4) Reported data either did not permit any further quantification or did not enable us to calculate the coefficient of lead in a multiple regression model.

Some studies were excluded on the basis of more than one of the above criteria. The first criterion effectively excludes most of the early studies in this area since these simply compared high- and low-lead groups, with limited or no control for relevant covariates. The second criterion was selected to avoid overcontrol. The one study that was excluded on this basis also involved a very small sample (multiple regression with 17 covariates and complete data on 48 subjects).

Two of the studies originally analyzed the data by dichotomizing lead exposure. The data were later reanalyzed by regression, treating exposure as a continuous variable. We used the results reported in the reanalyses. Supplementary information about the regression analysis was obtained from the authors of two studies.

Data Analysis

To achieve an acceptable level of homogeneity, the studies were divided into two groups according to the type of tissue analyzed for lead (blood or tooth). The *P* values within each group were compared for homogeneity using the technique of Rosenthal, which is based on the sum of the squared deviations of the *t* values for lead from the group mean.

Joint *P* values for lead were calculated for each of the two groups using two different approaches proposed by Fisher and by Mosteller and Bush. In Fisher's procedure, the logarithm of the product of the individual *P* values is multiplied by −2. The resulting quantity has a χ^2 distribution with 2N *df*. In the procedure by Mosteller and Bush, the weighted sum of the *t* values of the lead coefficient is computed, with each coefficient being weighted by its *df*. This method effectively weights each study by the number of subjects involved. It is particularly useful in this meta-analysis because of the wide range of sample sizes (75 to 724).

For each study, the partial correlation coefficient of lead was derived from the corresponding *t* value and was used as a measure of effect size. These coefficients were transformed to *z* scores using Fisher's transformation and were then compared via a χ^2 statistic. When the hypothesis of homogeneity was not rejected, the values of partial *r* from each study were treated as independent estimates of a common (group) partial correlation. Weighted *z* score averages were computed and were used to construct 95% confidence intervals

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Table 2.—Studies Included in the Meta-analysis*

Study	Year	Exposure Measure	Outcome Measure	Publication Status	Subjects' Age, y	Country
Yule et al	1981	Blood	WISC-R V, F	Journal	6-12	United Kingdom
Lansdown et al	1986	Blood	WISC-R V, F	Journal	Preschool	United Kingdom
Winneke et al	1983	Tooth	WISC-R V, F	Journal	7-12	Germany
Needleman et al	1985	Tooth	WISC-R V, F	Journal	7-8	United States
Ernhart et al	1985	Blood	McCarthy Scale	Journal	Preschool	United States
Schroeder et al	1985	Blood	Bayley/Stanford Binet IQ Scale	Journal	1-6	United States
Hawk et al	1986	Blood	Stanford Binet IQ Scale	Journal	3-7	United States
Fergusson et al	1987	Tooth	WISC-R V, F	Journal	8-9	New Zealand
Fulton et al	1987	Blood	British Ability Scale C	Journal	6-9	United Kingdom
Hatzakis et al	1987	Blood	WISC-R V, F	PROC	7-12	Greece
Pocock et al	1987	Tooth	WISC-R F	Journal	6	United Kingdom
Hansen et al	1987	Tooth	WISC-R V, F	PROC	7-8	Denmark

*WISC-R indicates Wechsler Intelligence Scale for Children–Revised; V, verbal; F, full-scale; and PROC, proceedings of meeting.

Table 3.—Covariates Entered Into the Final Multiple Regression Model*

Study‡	SES	Perinatal Factors	Postnatal Factors	Physical Factors	Gender	Parent IQ	Parental Rearing	Lead Coefficients†	
								Unadjusted	Final Model
Yule et al (2)	*			Age				NA	-8.08 (4.60)
Lansdown et al (2)	*			Age				NA	2.15 (4.49)
Winneke et al (52)	*		*	*	*			NA	-0.125 ([illegible])
Needleman et al (5)	*	*				*		NA	-0.21 (0.07)
Ernhart et al (3)		*	*	Age		*		NA	NA
Schroeder et al (7)	*							NA	-0.198 (0.07)
Hawk et al (1)	*				*	*	*	-0.488	-0.256 (0.15)
Fergusson et al (7)	*	*	*		*			NA	-1.48 (1.25)
Fulton et al (21)§	*	*		*	*	*	*	-5.45 (1.5)	-3.70 (1.31)
Hatzakis et al (10)	*	*				*	*	-0.378	-0.286 (0.07)
Smith et al (18)	*	*	*		*	*	*	-2.86 (0.86)	-0.77 (0.63)
Hansen et al (6)	*		*					NA	-4.27 (1.21)

*Asterisk (*) indicates those factors entered into the model, and SES, socioeconomic status.
†NA indicates not available. Where available, coefficients for lead are given for the unadjusted bivariate model and the final multivariate model.
‡The number of coefficients entered into the initial model is in parentheses.
§The SE of the coefficients was estimated from the data.

for the group partial correlation coefficient.

Power for each study to find a "small" effect was computed using the method (and program) described in Gatsonis and Sampson. We used the definition by Cohen of a "small" effect (partial r = .14).

Finally, to assess whether the exclusion of 12 of the original 24 studies had a biasing effect on our conclusions, we used Fisher's aggregation technique in an analysis that included all 24 studies. For most of the early studies, *P* values were either given in the published reports or derived on the basis of the published data. In the few cases where a *P* value was not available, we followed a conservative approach and assumed it was .5.

RESULTS

All studies considered and reasons for exclusion are listed in Table 1. Of the 12 excluded studies, 5 reported an effect significant at the .05 level and 7 did not. Twelve studies were included in the meta-analysis; 7 of them measured exposure by blood lead and 5 by tooth lead values (Table 2). The two groups were analyzed separately. In 11 of the 12 studies reviewed, the *t* value of the regression coefficient for lead was negative, ranging from −3.86 to 0.48 in the blood lead group and from −3.0 to −0.08 in the tooth lead group. The partial correlation coefficient of lead ranged from −.27 to .06 and from −.2 to −.003, respectively, for the two groups.

The dependent variable (IQ) was measured by the Wechsler Intelligence Scale for Children–Revised in eight studies. Two studies employed the Stanford Binet IQ Scale, one employed the British Ability Scale, and one employed the McCarthy Scale. The comparison of the distributions of lead exposure was hindered by two difficulties: (1) methods for measuring lead level differed, particularly in the tooth lead group, and (2) summary descriptions of the distribution of lead exposure also differed. In the blood lead group, the lead exposure in the study by Lansdown et al (mean, 0.62 μmol/L) was among the lowest, while the exposure in the study by Schroeder et al (median, 1.46 μmol/L) was among the highest. In the tooth lead group, where analytic methods were different, the lead exposure in the study by Smith et al was among the lowest (248 of 402 children had tooth lead concentration <5.5 ppm), while the exposure in the study by Needleman et al (mean, 12.7 ppm) was among the highest. The sets of covariates included in the regression equations differed for each study, although most covariates purported to measure factors that were similar across studies. It is impractical to present herein a detailed list of the covariates for each study. A condensed form of this information is in Table 3, in which we classified the various covariates into groups on the basis of seven factors. Where available, the unadjusted coefficient of lead is also included in Table 3, along with the coefficient of lead in the final model. In some studies the logarithm of the lead measurement was used in the regression equations.

The *P* values for the common direc-

Table 4.—Results of Synthesis of 12 Studies

Study	Weighted *t* Values: *t*	Weighted *t* Values: *P* (One-Sided)	Fisher's Technique: χ^2	Fisher's Technique: *P*
Blood Lead Studies				
All studies	−5.46	<.0001	61.20	<.0001
Eliminating one study at a time (study eliminated)				
Hatzakis et al	−5.09	<.0001	42.87	<.0001
Hawk et al	−5.34	<.0001	56.3	<.0001
Schroeder et al	−5.15	<.0001	46.96	<.0001
Fulton et al	−4.67	<.0001	46.08	<.0001
Yule et al	−5.26	<.0001	54.86	<.0001
Lansdown et al	−5.58	<.0001	60.52	<.0001
Ernhart et al	−5.31	<.0001	54.86	<.0001
Combining studies using log-transformed values (Fulton et al, Yule et al, and Lansdown et al)			16.83	.006
Tooth Lead Studies				
All studies	−2.66	.004	33.11	<.0005
Eliminating one study at a time (study eliminated)				
Needleman et al	−1.97	.024	19.29	<.025
Hansen et al	−2.3	.011	23.9	<.005
Winneke et al	−2.67	.004	31.08	<.0005
Smith et al	−2.39	.009	28.80	<.0005
Ferguson et al	−3.04	.001	28.86	<.0005
Combining studies using log-transformed values (Smith et al and Ferguson et al)	−1.61	.001	8.08	<.0005

Table 5.—Lead Coefficients for Full-scale IQ Scores*

Study	Coefficient	SE	*t*	*P* (One-Sided)	Sample Size	Partial *r*	Total R^2
Blood Lead Studies							
Hatzakis et al	−0.27	0.071†	−3.861†	.0001	509	−.17	0.25
Hawk et al	−0.25	0.15	−1.67	.05	75	−.20	0.21
Schroeder et al	−0.2	0.071†	−2.78	.003	104	−.27	NA
Fulton et al‡	−3.7	1.37	−2.77	.003	501	−.12	0.46
Yule et al‡	−8.08	4.63	−1.75	.04	129	−.16	NA
Lansdown et al‡	2.15	4.48†	0.48	.68	86	.05	NA
Ernhart et al	NA	NA	−1.81	.04	80	−.20	NA
(Average weighted partial *r* = −.152; 95% confidence interval, −.2 to −.1)							
Tooth Lead Studies							
Needleman et al	−0.21	0.07	−3	.001	218	−.20	0.35
Hansen et al	−4.27	1.91	−2.23§	.01	156	−.18	0.2
Winneke et al	−0.13	4.66	−0.03§	.49	115	−.003	0.13
Pocock et al‡	−0.77	0.63	−1.22	.11	388	−.06	NA
Ferguson et al‡	−1.46	1.25	−1.17	.12	724	−.04	NA
Average weighted partial *r* = −.08; 95% confidence interval, −.13 to −.03)							

*NA indicates not available.
†Estimated from data in article.
‡Log transforms.
§Obtained from the author.

tional hypothesis that lead is negatively correlated with IQ were tabulated. Before combining the probabilities, the homogeneity of the *P* values was assessed. The χ^2 statistics were 11.02 ($df=6$, $P=.09$) and 5.13 ($df=4$, $P=.26$) for the blood lead and tooth lead group, respectively. Thus, the hypothesis of homogeneity cannot be rejected for either group.

Combined *P* values in the blood lead group were less than .0001 for both methods of combining probabilities. The corresponding combined *P* values for the tooth lead group were less than .0005 and .004, respectively.

Sensitivity Analysis

The sensitivity of the findings was examined by removing the studies one by one from the analysis and recalculating combined *P* values (Table 4). For the tooth lead group the highest combined *P* value was .025 and the lowest was .0001. The corresponding figures for the blood lead group were below .0001. The overall finding of a significant lead effect is supported by both methods of combining the data. No single study seems to be responsible for the significance of the final finding.

Effect Size

In the case of multiple regression/correlation studies, the usual measure of effect size is the partial correlation coefficient (partial *r*).[illegible] Derived partial *r*'s for the 12 studies under review are given in Table 5.

Each partial *r* was converted to a *z* score using Fisher's *z* transform. The χ^2 statistics for homogeneity were 5.78 ($df=6$, $P>.4$) for the blood lead group and 6.44 ($df=4$, $P>.1$) for the tooth lead group. The hypothesis of homogeneity of the effect sizes cannot be rejected for either of the two groups. The weighted *z* score averages were −.152 (SE = .027) and −.08 (SE = .025), respectively. In the original scale, the approximate 95% confidence intervals for the group partial *r* were −.15 ± .05 for the blood lead group and −.08 ± .05 for the tooth lead group.

The results of the analysis in terms of the partial *r*'s support those obtained from the analysis of the *P* values. Neither approach provides an overall estimate of the raw effect size, ie, of the average change in IQ units per unit change in lead exposure. A meaningful attempt to arrive at such an overall estimate is precluded by the substantial differences in model specification among the studies, as well as in units and methods of measuring lead exposure and outcome.

Selection Bias and the File Drawer Problem

There were two basic steps in the selection of studies for this meta-analysis: (1) the retrieval of studies and (2) the formulation and application of exclusion criteria to the retrieved studies. The possibility of bias in both steps was investigated. In particular with respect to the second step, calculations with all the original 24 studies included showed that Fisher's statistic was 93.8 ($df=34$, $P<.0001$) for the blood lead group, 42.5 ($df=14$, $P<.001$) for the tooth lead group, and 136.4 ($df=48$, $P<.0001$) for all studies together. This is evidence that the application of the exclusion criteria was not an important source of bias in this meta-analysis.

The possibility of bias resulting from the first step has been termed the *file drawer problem*.[illegible] Such bias may result from at least two sources (beyond faults in the retrieval process): the failure of all investigators to report their results or the failure of journals to publish all results submitted. Studies that show a statistically significant result do tend to be published more frequently.

We estimated the magnitude of the file drawer problem by calculating the number of unpublished nonsignificant

Table 6.—Power Calculations for "Small" Effects of Lead (α = .05; Partial r = .14)

Study	Sample Size	No. of Covariates (Final)	Power
	Blood Lead Studies		
Fulton et al	501	14	0.87
Hatzakis et al	500	8	0.86
Hawk et al	75	5	0.21
Schroeder et al	104	7	0.28
Yule et al	129	2	0.35
Lansdown et al	80	2	0.25
Ernhart et al	46	4	0.20
	Tooth Lead Studies		
Needleman et al	218	5	0.63
Fergusson et al	724	8	0.98
Smith et al	388	10	0.78
Winneke et al	115	4	0.31
Hansen et al	156	7	0.40

studies that would be necessary to bring the overall *P* value to greater than .05. Using the procedure of Rosenthal, we found that 26 null result studies would be necessary to dilute the finding for the tooth lead group and that 67 would be necessary for the blood lead group. This procedure assumes that the mean *z* score of the unseen studies is 0. A more stringent procedure is suggested by Iyengar and Greenhouse, which assumes that all unseen studies simply are not significant at the .05 level. Under this assumption, it would require 16 and 35 studies to dilute the finding for the tooth lead group and the blood lead group, respectively. Given the expense of conducting human studies of lead exposure and the amount of attention directed to this question, it is unlikely that this number of negative studies have escaped notice.

Power Calculations

The studies included in this meta-analysis are observational. The values of the covariates cannot be fixed in advance by design but are themselves outcomes of the study. Any calculation of power must account for this extra variability. Table 6 presents the a priori power of each study to detect a partial *r* of .14 (denoted as a "small" effect). "Small" in this sense does not mean biologically unimportant, it means difficult to identify. Cohen has pointed out that a result of this size "all too frequently in practice represents the true order of magnitude of the effects being tested." As can be seen from Table 5, a partial *r* equal to −.14 is near the center of the values for the partial correlation coefficient that were derived from the studies under review. Of the 12 studies, 8 had power below 60% to detect an effect of this magnitude.

The power figures given here are optimistic: they are calculated on the number of covariates present in the final model reported in each study. Most studies, however, initially controlled for many more covariates than those in the final model. As few articles gave information about missing values in the data, it is possible that some of the sample sizes used to calculate power are larger than the effective sample sizes of the studies.

Some Methodological Issues

The inclusion criteria ensured that the studies analyzed provided an acceptable minimum control for relevant covariates. In two studies, control was done only for social class. Multiple regression analysis was employed in all studies, usually in stepwise form. No study reported any analysis of residuals, model checking, and detection of possible outliers in the data. Only two studies attempted to select an "optimal" regression model in a formal way. No study addressed the issue of errors in measurement of the independent variables. The question of errors in variables is particularly relevant when measuring exposure at low levels. Other covariates that represent arbitrary constructs (eg, marital relationships, parental interest, parental involvement in school, and so on) are also particularly vulnerable to errors-in-variables problems.

COMMENT

The overall evidence from our meta-analysis establishes a strong link between low-dose lead exposure and intellectual deficit in children. A natural question that arises at this point is whether the link is a causal one. The answer to this question goes beyond the formal meta-analytic method. Some of the epistemological issues encountered in making causal inferences are discussed below.

The effects of lead on the central nervous system are embedded in a complex process involving biologic, environmental, familial, and socioeconomic factors. Epidemiologic studies cannot, by themselves, establish causal relationships. Causality is not subject to empirical proof, whether in the field or in the laboratory. Given that direct demonstration of proof of a low-dose lead effect in a naturalistic setting is not achievable, epidemiologists rely on canons that, if satisfied, permit the conservative drawing of causal inferences. They are (1) time precedence of the putative cause, (2) biologic plausibility, (3) nonspuriousness, and (4) consistency.

Cross-sectional studies such as those reviewed herein cannot establish the time precedence of lead exposure; the level of lead was measured at the same time as IQ. The claim has been made that neurobehavioral deficits result in excess lead intake, ie, deficient children mouth more leaded substances. This assertion has been effectively refuted by forward studies of lead exposure beginning at birth. These studies have shown a clear relationship between umbilical cord blood lead levels and later development at 6 to 24 months.

Biologic plausibility demands that mechanisms at a lower biologic level have been demonstrated to explain the phenomenon under examination. Lead is a thoroughly investigated neurotoxin. Among many effects that have been demonstrated, lead has been shown to affect neurotransmitter activity, brain adenyl cyclase activity, and dendritic complexity. Demonstration of dose-response relationships strengthens the plausibility of the relationships studied. Convincing demonstrations of dose-related behavioral effects have been made in animal studies. Epidemiologic demonstrations of association between dose (blood or tooth lead levels) and response (teachers' ratings of classroom behavior and reaction time under varying intervals of delay) also have been published.

Nonspuriousness means that the relationship put forth in the causal claim is not due to a confounder or a set of confounders. Complete confounder control is impossible in real world studies. In most studies reviewed, control of confounders has reduced the magnitude of the lead-IQ effect but has not obliterated it. The argument for nonspuriousness is further strengthened by the evidence provided by animal studies in rodents and subhuman primates, which produced cognate outcomes in cross-fostered litter mates.

Finally, consistency requires that the phenomenon be demonstrated in different studies under similar but not identical circumstances. The statistical nature of these investigations requires an extended notion of consistency. Even if the effect under study exists in nature, the *P* values and effect sizes reported in investigations of the question will vary in magnitude, and not all studies will give a significant result.

A different type of evidence for consistency was offered in the study by Wallsten and Whitfield. This evidence is based on the probabilistically encoded opinions of six lead experts of widely ranging viewpoints about the dose-response relation between lead exposure and IQ. Five of the six experts' estimates of the dose-response curve were convergent, leading the authors to state: "In view of the extensive debate concerning the effects of lead on IQ, the degree of consensus reflected in the

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study's results is notable, especially since the experts were selected so as to span the full range of opinion.[47]

The four previously cited reviews of the studies of lead at low dose differed in their evaluation of essentially the same evidence. One review came to a qualified negative conclusion,[2] one came to a positive conclusion,[3] and two found the evidence inconclusive.[4,5] This difference of opinion partly proceeds from a limitation inherent in the method of narrative reviewing; it essentially evaluates each study in isolation and is unable to achieve a systematic synthesis. Meta-analysis avoids this limitation and includes all studies in a joint inference. Using this method, and incorporating into our review a number of recent studies that were not available to the earlier reviewers, we found that although the sample of studies varied widely in their individual power to find an effect, and not all found an effect by the conventional rule of $P<.05$, 11 of 12 studies reviewed reported a negative coefficient for lead. The joint probability of the findings reported occurring by chance under the null hypothesis was quite small, and this was not materially influenced by any single study. The estimated effect sizes for the two groups were both significantly different from zero. These findings, taken in sum, permit a strong inference that low-dose lead exposure is causally associated with deficits in psychometric intelligence.

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THE LONG-TERM EFFECTS OF EXPOSURE TO LOW DOSES OF LEAD IN CHILDHOOD

An 11-Year Follow-up Report

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Abstract To determine whether the effects of low-level lead exposure persist, we reexamined 132 of 270 young adults who had initially been studied as primary schoolchildren in 1975 through 1978. In the earlier study, neurobehavioral functioning was found to be inversely related to dentin lead levels. As compared with those we restudied, the other 138 subjects had had somewhat higher lead levels on earlier analysis, as well as significantly lower IQ scores and poorer teachers' ratings of classroom behavior.

When the 132 subjects were reexamined in 1988, impairment in neurobehavioral function was still found to be related to the lead content of teeth shed at the ages of six and seven. The young people with dentin lead levels >20 ppm had a markedly higher risk of dropping out of high school (adjusted odds ratio, 7.4; 95 percent confidence interval, 1.4 to 40.7) and of having a reading disability (odds ratio, 5.8; 95 percent confidence interval, 1.7 to 19.7) as compared with those with dentin lead levels <10 ppm. Higher lead levels in childhood were also significantly associated with lower class standing in high school, increased absenteeism, lower vocabulary and grammatical-reasoning scores, poorer hand–eye coordination, longer reaction times, and slower finger tapping. No significant associations were found with the results of 10 other tests of neurobehavioral functioning. Lead levels were inversely related to self-reports of minor delinquent activity.

We conclude that exposure to lead in childhood is associated with deficits in central nervous system functioning that persist into young adulthood. (N Engl J Med 1990; 322:83-8.)

WITHIN the past three years, the Environmental Protection Agency and the Agency for Toxic Substances and Disease Registry have concluded in policy statements that lead at low doses is a serious threat to the central nervous systems of infants and children.[1,2] These policy statements have been based on a growing convergence of results from both epidemiologic and experimental studies of lead toxicity in the United States, Europe, and Australia.[3-8] Whether the effects on the central nervous system of exposure to low doses of lead that have been observed in infants and children persist has received limited attention. Only three follow-up studies have been published to date, and the longest follow-up has been five years.[9-11] No data have yet been reported on whether early disturbances influence functional abilities in later life.

In 1979 we reported that first- and second-grade children without symptoms of plumbism, but with elevated dentin lead levels, had deficits in psychometric intelligence scores, speech and language processing, attention, and classroom performance.[5] When they were studied in the fifth grade, the children with high dentin lead levels had lower IQ scores, needed more special academic services, and had a significantly higher rate of failure in school than other children.[9] We have now evaluated the neuropsychological and academic performance in young adulthood of 132 of the original sample of 270 subjects, and we report the relation of their recent performance to their exposure to lead, as measured 11 years earlier.

Methods

Sample

The initial sample was chosen from the population of 3329 children enrolled in the first and second grades in the Chelsea and Somerville, Massachusetts, school systems between 1975 and 1978. Of this population, 70 percent provided at least one of their shed primary teeth for lead analysis. From this sample of 2335 children, 97 percent of whom were white, we identified 270 from English-speaking homes whose initial dentin lead levels were either >24 ppm or <6 ppm. These children (mean age, 7.3 years) underwent an extensive neurobehavioral examination. More teeth were subsequently collected and analyzed, and the subjects whose teeth were discordant with respect to lead level according to a priori criteria were excluded from the data analysis. Also excluded from the analysis were children who had not been discharged from the hospital after birth at the same time as their mothers, who had a noteworthy head injury, or who were reported to have had plumbism.[5]

In a later reanalysis, conducted in response to suggestions from the Environmental Protection Agency,[12] the tooth lead level was treated as a continuous variable. A mean dentin lead level was computed for each subject from all the teeth collected. The exclusionary factors previously used were found not to be related to outcome scores. The subjects initially excluded were therefore not excluded from this follow-up sample.

The 270 subjects tested from 1975 to 1978 constitute the base population for this report. From old research records, telephone directories, town records, and driver's-license rolls, we located 177 subjects. Of these, 132 agreed to participate, and the remaining 45 declined. The subjects were paid $35 each and received travel expenses. Ten subjects tested in 1988 had been excluded from the analysis reported in 1979 because their parents stated at the time of testing that the children had elevated blood lead levels or had undergone chelation for lead poisoning. This group is discussed separately in this report. The mean age of the 132 subjects at the 1988 reexamination was 18.4 years; the mean length of time between the two examinations was 11.1 years. All but four subjects in the current follow-up study were white. No clinical manifestations of lead exposure were recorded in the earlier interviews for the 122 subjects who were not treated with chelating agents.

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The research protocol and informed-consent procedures were approved by the institutional review boards of the Children's Hospital of Pittsburgh and the Children's Hospital, Boston. Informed consent was given by all the subjects or their parents.

Classification of Lead Exposure

All the dentin lead levels measured from 1975 through 1977 were used to compute an arithmetic mean lead concentration for each subject. The lead burden was treated in two ways: as an interval variable in linear regressions and as a categorical variable — i.e., high (>20 ppm), medium (10 to 19.9 ppm), and low (<10 ppm) — in the logistic regressions described below. Lead levels in venous blood were measured at the time of the reexamination to estimate current exposure. This practice was discontinued after the first 48 subjects were tested, because none had a lead level exceeding 0.34 μmol per liter (7 μg per deciliter), well below the Centers for Disease Control's definition of undue lead exposure of 1.25 μmol per liter (25 μg per deciliter).

Behavioral Evaluation

The subjects were evaluated individually by a single examiner, who remained blinded to their lead-exposure status until all the data had been coded and entered into a computer data base. All assessments were carried out in a fixed order; the duration of the testing was about two hours.

Neurobehavioral Evaluation System

The subjects completed an automated assessment battery in which they used a personal computer, joystick, and response key.[13] We selected the following items from the battery for evaluation:

Continuous-performance test.[14]

Symbol-digit substitution, an adaptation of the Wechsler item for computer administration.

Hand-eye coordination. Using a joystick to move the cursor, the subject traced over a large sine wave generated on the monitor screen; deviations from the line (root mean square error) were recorded.

Simple visual-reaction time. Subjects pressed the response key when an O appeared on the screen; the interval before the stimulus was varied randomly.

Finger tapping. The subject pressed a response button as many times as possible during a 10-second period; both hands were tested.

Pattern memory. The subject was presented with a computer-generated pattern formed by a 10-by-10 array of dark and bright elements. After a brief exposure, the subject was presented with three patterns, only one of which was identical to the original pattern. The number of correct responses and the length of time to the correct choice were recorded.

Pattern comparison. The subject was presented with three computer-generated patterns on the 10-by-10 array. Two were identical, and one differed slightly from the other two. The subject was required to select the nonmatching pattern.

Serial-digit learning. The subject was presented with a string of 10 digits, then asked to enter the string into the computer. After an error, the same stimulus was presented, and the second trial began.

Vocabulary. For each of 25 words, the subject chose the word most nearly synonymous from a list of four choices.

Grammatical reasoning. The subject was presented with a pair of letters, A and B, whose relative position varied. Then the screen cleared, and the letters were replaced by a sentence that described the order of the letters. The sentence might be active or passive, affirmative or negative, true or false (examples are "A follows B" and "B is not followed by A"). The subject had to choose the correct sentences, and the number of errors was recorded.

Switching attention. The subject was required to choose which key to press in response to three different instructions. In the "side" trials, the subject had to press the key on the same side as the stimulus. In the "direction" trials, the correct choice was the direction in which an arrow pointed. Before each trial in the third set, the subject was told whether to choose the side the arrow was on or the direction in which it pointed.

Mood scales. This test was derived from the Profile of Mood States.[15] Five scores were computed for tension, anger, depression, fatigue, and confusion.

The following tests were also used to evaluate neurobehavioral functioning:

California Verbal Learning Test

The California Verbal Learning Test[16] was used to assess multiple strategies and processes involved in verbal learning and memory. Scores for immediate and delayed recall were also obtained.

Boston Naming Test

In the Boston Naming Test,[17] the subject was presented with 60 pictures in order of increasing difficulty and asked to name the objects shown.

Rey–Osterreith Complex Figure Test

The Rey–Osterreith Complex Figure Test[18] was used to evaluate visual-motor and visual-spatial skills. The subject was asked to copy an abstract geometric figure and then to draw it from memory both immediately and after 30 minutes. Accuracy and organization scores were calculated.

Word-Identification Test

Form B from the Woodcock Reading Mastery Test was used to evaluate reading skill. Grade-equivalency scores were calculated from raw scores. Reading disability was defined as indicated by scores two grade levels below the score expected on the basis of the highest grade completed.

Self-Reports of Delinquency

The subjects completed a structured questionnaire from the National Youth Survey[19] that included scales for minor antisocial behavior and for violent crimes.

Review of School Records

High-school records were obtained for all but two of the subjects tested. Class size and rank, the highest grade completed, and the number of days absent and tardy in the last full semester were recorded. Students who were still in the 11th grade at the time of testing were not included in analyses of the highest grade completed. Class rank was computed as 1 − (class rank/class size).

Statistical Analysis

To evaluate whether the participants in this follow-up evaluation were representative of the original cohort, subjects who were tested and not tested in 1988 were compared in terms of variables reported in 1979, including dentin lead levels, covariates not related to lead exposure, teachers' ratings of classroom behavior, and IQ scores. In addition, we carried out separate regressions of dentin lead level against IQ score as measured between 1976 and 1978 for subjects tested and not tested in 1988. We then performed a regression on both groups taken together, entering both a dummy term for participation in the current follow-up (yes or no) and a lead-level-by-participation status term.

To evaluate the relation between early exposure to lead and each of the continuously distributed outcome variables, subjects were classified according to dentin lead-level quartiles, and mean scores, adjusted for covariates, were computed. Ordinary least-squares lin-

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[illegible] regression, with the mean or log-mean dentin lead level as the main effect, was used to estimate the significance of the relation. Outcomes that were significantly associated with lead exposure in these bivariate analyses were further evaluated by multiple regression analysis. Ten covariates were included in the model. They were the mother's age at the time of the subject's birth, the mother's educational level, the mother's IQ, family size, socioeconomic status (a two-factor Hollingshead index), sex, age at the time of testing, birth order, alcohol use, and whether the subject and the mother left the hospital together after the subject's birth. The lead measure (the mean or the log of the mean) that produced the best-fitted model (highest R^2) is reported. Five of these covariates were employed in the first study of these subjects and shown to be influential. Five others (sex, age at testing, prolonged hospitalization as a neonate, birth order, and current alcohol use) were added to the model on the basis of prior knowledge of their effects on psychometric function. Logistic-regression analysis was used to model the association of lead level and two outcomes treated categorically (failure to graduate from high school and reading disability). In this analysis, we controlled for the covariates listed above. Two indicator variables were used to represent the three exposure groups. Odds ratios and 95 percent confidence intervals, adjusted for covariates, were computed for the high-lead-level group, with the low-lead-level group used as the reference group.

RESULTS

Selection Bias

The 132 subjects who were retested in 1988 (Table 1) were not representative of the group of 270 subjects tested in 1979. The subjects we retested tended to have slightly lower dentin lead levels, more highly educated families of higher socioeconomic status, and mothers with higher IQs and better obstetrical histories; a higher proportion of the retested subjects were girls. In addition, they had had fewer head injuries and had significantly higher IQ scores and better teachers' ratings as reported in 1979. The slope of the regression of childhood IQ on dentin lead level was steeper in the group not tested in the follow-up study, although the difference from the slope in the group we retested was not statistically significant (F = 1.82, 1,196 df, P = 0.18).

Table 1. Comparison of Subjects Tested and Not Tested in 1988.*

Characteristic	Tested (N = 132)	Not Tested (N = 138)	P Value
Lead-level group (%)			
Low	50	47.8	—
Middle	22.7	16.7	—
High	27.3	35.5	0.39†
Birth order	2.3±1.6	3.0±1.9	0.016
No. of live births	2.8±1.5	3.2±1.6	0.09
Father's education (yr)	12.2±2.6	11.4±2.0	0.009
Mother's education (yr)	12.0±2.2	11.1±2.1	0.0005
Mother's IQ	112±15	108±15	0.017
Mother's age at subject's birth (yr)	25.5±5.0	25.1±5.8	0.7
Father's age at subject's birth (yr)	28.3±7.8	28.8±7.9	0.6
Gestation (wk)	39.9±2.0	40.0±1.7	0.7
Birth weight (g)	3376±608	3312±600	0.40
Sex (%)			
Female	55.3	42.8	
Male	44.7	57.2	0.04
Head injuries (%)	3.8	8.7	0.09
Teachers' ratings (1979 sum score)	9.1±2.8	8.2±3.6	0.024
Full-scale IQ (1979)	107.5±14	99.5±15	0.001

*Plus–minus values are means ±SD. †By chi-square test for all lead-level groups.

Academic and Neurobehavioral Outcomes

Table 2 shows the covariate-adjusted scores of the 122 subjects who did not have clinical plumbism, according to their dentin lead concentrations. Table 3 summarizes the results of modeling the relation between early exposure to lead and outcome by multiple regression. Earlier exposure to lead was significantly associated with diminished academic success. Among children with dentin lead levels >20 ppm, as compared with those whose dentin lead levels were <10 ppm, the unadjusted odds ratio for failure to graduate from high school was 4.6 (95 percent confidence interval, 1.2 to 17.4). Adjustment for covariates increased the odds ratio to 7.4 (95 percent confidence interval, 1.4 to 40.8). Higher dentin lead levels were also associated with lower class rank, increased absenteeism, lower scores on vocabulary and grammatical-reasoning tests, significantly slower finger-tapping speed, longer reaction times, poorer hand-eye coordination, and lower reading scores. In subjects with dentin lead levels >20 ppm, the unadjusted odds ratio for having a reading disability, defined by a score two grades below that expected for the highest grade completed, was 3.9 (95 percent confi-

Table 2. Outcomes in Young Adulthood According to Dentin Lead Concentration in Childhood.*

Outcome Variable	Lead Concentration			
	Lowest (<5.9 ppm)	Low (6.0–9.1 ppm)	High (9.2–21.1 ppm)	Highest (>21.1 ppm)
No. of subjects	30	31	30	31
Reading score (words read correctly)	143.8	142.7	140.2	135.2
Reading grade equivalent (grade level)	12.2	11.9	11.2	10.1
Highest grade achieved (grade level)	11.7	11.9	11.5	11.3
Class standing (percentile)	0.60	0.59	0.48	0.45
Absence from school (no. of days/semester)	12.0	12.0	17.9	20.8
Vocabulary (words correct)	18.0	16.4	17.6	14.6
Grammatical reasoning (no. incorrect)	13.1	13.0	12.8	15.8
Hand-eye coordination†	5.1	5.4	5.5	6.2
Reaction time (msec)				
Preferred hand	246.6	255.1	267.3	275.1
Nonpreferred hand	241.2	258.2	258.4	261.2
Finger tapping (no./10 sec)	46.6	47.2	45.0	43.9

*The subjects were divided into groups according to lead-level quartiles. The values shown are least-square mean scores after adjustment for covariates. Subjects with clinical plumbism have been excluded.

†For hand-eye coordination, larger numbers indicate more errors.

Table 3. Regression of Outcomes in Young Adulthood on Dentin Lead Levels in Childhood.*

Outcome Variable	Univariate Regression				Multiple Regression			
	R^2	Parameter Estimate	SE	P Value	R^2	Parameter Estimate	SE	P Value
Highest grade achieved	0.061	−0.027	0.009	0.006	0.119	−0.027	0.01	0.013
Reading grade equivalent	0.121	−0.07	0.018	0.0001	0.230	−0.073	0.021	0.001
Class standing	0.079	−0.006	0.003	0.008	0.248	−0.004	0.002	0.048
Absence from school†	0.071	4.8	1.7	0.006	0.209	4.73	1.8	0.01
Grammatical reasoning	0.051	0.139	0.062	0.013	0.197	0.176	0.068	0.011
Vocabulary	0.108	−0.130	0.037	0.0009	0.334	−0.122	0.033	0.001
Finger tapping	0.031	−0.104	0.05	0.05	0.336	−0.133	0.05	0.01
Hand–eye coordination	0.040	0.041	0.018	0.02	0.195	0.048	0.019	0.01
Reaction time†								
Preferred hand	0.025	11.8	6.66	0.08	0.242	12.9	6.3	0.042
Nonpreferred hand	0.03	11.3	6.0	0.06	0.220	10.3	5.5	0.06
Minor antisocial behavior†	0.020	−0.639	0.36	0.082	0.300	−0.739	0.35	0.039

*The following covariates were controlled for in the multiple regression analysis: age, sex, birth order, family size, mother's age at the subject's birth, length of the neonatal stay in the hospital, mother's education level, mother's IQ, socioeconomic status, and current alcohol use.

†The natural log of the mean dentin lead level was used as the main effect.

dence interval, 1.5 to 10.5). Adjustment for covariates increased the odds ratio to 5.8 (95 percent confidence interval, 1.7 to 19.7). For most outcomes, neither the size of the lead regression coefficients nor their standard errors were substantially changed by adjustment for covariates.

Of the 10 children with clinical plumbism (who either underwent chelation or were reported to have had elevated blood lead levels), 3 of 7 (43 percent) dropped out before graduating from high school (3 others are still in school), and 5 of 10 (50 percent) have reading disabilities. When the children with plumbism were grouped with the other subjects according to quartiles for dentin lead levels, a dose–response relation was evident for both outcomes (Fig. 1 and 2).

Early exposure to lead was not significantly associated with performance on the symbol-digit or serial-digit tests, the continuous-performance test, pattern memory or pattern comparison, switching attention, the California Verbal Learning Test, the Rey–Osterreith figures, the Boston Naming Test, or mood scores. The lead level was inversely related to the summed score on the self-report of delinquency questionnaire, which consisted primarily of reports of minor antisocial behavior.

When subjects were divided into two groups according to their dentin lead levels (<10 ppm vs. ≥10 ppm), high dentin lead levels predicted future failure to graduate from high school with a sensitivity (±SE) of 0.71±0.12 and a specificity of 0.61±0.05 (Table 4).

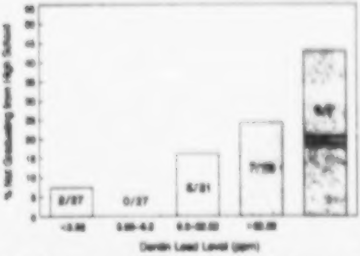

Figure 1. The Proportion of Subjects Who Did Not Graduate from High School, Classified According to Their Past Exposure to Lead.

Asymptomatic subjects are classified according to lead-level quartiles. Seven of the 10 subjects who were earlier reported to have clinical plumbism are shown in a separate column. No school records were found for two subjects. One subject was not tested but reported that she had graduated from high school. (There are therefore 121 subjects represented in this figure.) Ten subjects (three with reported plumbism and seven asymptomatic subjects) are still attending high school and are therefore not shown here. The numbers in each column indicate the number who did not graduate and the total number in the category.

Discussion

In this extended follow-up study, in which the mean length of follow-up was 11.1 years, we found that the associations reported earlier between lead and children's academic progress and cognitive functioning persisted into young adulthood. The persistent toxicity of lead was seen to result in significant and serious impairment of academic success, specifically a sevenfold increase in failure to graduate from high school, lower class standing, greater absenteeism, impairment of reading skills sufficiently extensive to be labeled reading disability (indicated by scores two grades below the expected scores), and deficits in vocabulary, fine motor skills, reaction time, and hand–eye coordination.

A number of issues require consideration when one is interpreting the data reported here. The first is the influence of selection bias on the associations we observed. The subjects retested in 1988 had more favorable characteristics than those who could not be located or who declined to participate. The subjects who were not retested tended to have had higher lead lev-

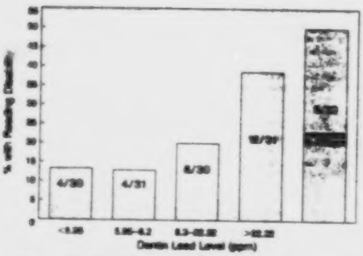

Figure 2. The Proportion of Subjects with Reading Disabilities, Classified According to Their Past Exposure to Lead.

Asymptomatic subjects are classified according to lead-level quartile, and 10 children with a history of clinical plumbism are shown separately. Reading disability is defined as indicated by a reading level two or more grades below the expected level. The numbers in each column indicate the number with a reading disability and the total number in the category.

els, lower socioeconomic status, and lower IQ scores and teachers' ratings of classroom behavior. The inverse relation between dentin lead levels and IQ reported in 1979 was stronger for the subjects who were not retested in 1988 than for those we retested, although the difference did not reach statistical significance. This finding is in agreement with the observation, made by us and others, that children from families in lower socioeconomic groups are more vulnerable to the effects of lead than children from more favored economic backgrounds.[30] We infer that the estimates made on the basis of the data on the 132 subjects we restudied are likely to be conservative. Indeed, had all the original subjects been located and retested, the magnitude of the effect of lead exposure might have been even greater.

Is the nature of the relation between lead and later outcome causal, or does it result from confounding by other variables? The association between lead and outcome reported here meets six criteria for valid causal inference: proper temporal sequence, strength of association, presence of a biologic gradient, nonspuriousness, consistency, and biologic plausibility.[31]

In this study, the exposure to lead preceded the school failure and the reading disabilities measured. The strength of the association, as measured by adjusted odds ratios of 7.4 and 5.8, was substantial. A dose–response relation has been demonstrated between exposure and numerous outcome variables (Table 2, Fig. 1 and 2). "Nonspuriousness" indicates that the association observed is not due to confounding. In this analysis, we controlled for both the covariates that were identified in 1979 as potential confounders and others we suspected were important. The magnitude of the effect of lead was reduced only slightly, if at all, by this procedure. The zero-order correlation between socioeconomic status and dentin lead levels in this sample was not great (r = 0.04). Many covariates that were important contributors to performance in the early grades (e.g., the mother's IQ and the mother's educational level) had less effect on the subject's performance in young adulthood. The results, moreover, are consistent with those of several other studies by workers who have reported lead-associated deficits in reading[6,22,23] and early classroom behavior.[24,25] The lead-related deficits in IQ, speech and language processing, and attention reported in 1979 provide plausible mechanisms by which lead could impair performance in class and produce eventual failure. Similar effects on learning have been demonstrated in the experimental studies by Gilbert and Rice of subhuman primates.[7] In these investigations, rhesus monkeys, administered lead only in the first 100 days of life, had impairments in learning as adolescents. In adolescence, the mean blood lead level of these monkeys was 0.73 μmol per liter (15 μg per deciliter).

The value accepted as the threshold for lead-engendered neurotoxicity in children has declined steadily over the past decade as more sophisticated population studies, with larger samples, better designs, and better analyses, have been conducted.[4,5,11,22,24,26-29] When this study was begun in 1975, the toxic level of lead in the blood was defined by the Centers for Disease Control as 2.0 μmol per liter (40 μg per deciliter). In 1973, the mean blood lead level in a subsample of 23 children chosen from among those with the highest dentin lead levels in an earlier study was 1.7 μmol per liter (34 μg per deciliter).[3] None of our subjects were symptomatic. That these subjects were exposed to high doses of lead after the original study was completed is unlikely. Lead exposure, the incidence of pica, and hand-to-mouth behavior diminish after the fifth year of life. The low blood lead levels found in these subjects in young adulthood (all <0.034 μmol per liter) provide convincing evidence that their later exposure to lead was not excessive.

The consensus on what level of lead is toxic has changed in recent years. After reviewing the studies published up to 1987, the Agency for Toxic Substances and Disease Registry defined the threshold for neurobehavioral toxicity as 0.5 to 0.7 μmol per liter

Table 4. Sensitivity and Specificity of the Dentin Lead Level in Childhood as a Predictor of Failure to Graduate from High School.*

High-School Graduation	Lead Level	
	≥10 ppm	<10 ppm
No	10	4
Yes	39	61

Sensitivity = 10/(10+4) = 0.71

Specificity = 61/(61+39) = 0.61

*Of the 132 asymptomatic subjects studied, 7 subjects who were still attending school at the time of this analysis were excluded. One subject's school records were not found. Of the 132 subjects retested in 1988, the 10 with clinical plumbism have been excluded.

(10 to 15 μg per deciliter).[1] The agency estimated that 3 to 4 million American children have blood lead levels in excess of 0.7 μmol per liter. The mean blood level among our subjects with high tooth lead levels, estimated in 1979 from a limited lead-screening program, was 1.6 μmol per liter (34 μg per deciliter) (range, 0.87 to 2.6 μmol per liter [18 to 54 μg per deciliter]). For subjects with low tooth lead levels, it was 1.2 μmol per liter (24 μg per deciliter) (range, 0.58 to 1.7 μmol per liter [12 to 36 μg per deciliter]). Thus, the lead levels in the reference sample used in the calculation of the odds ratios for one high-lead-level group were relatively high by contemporary standards.

The data presented here indicate that exposure to lead, even in children who remain asymptomatic, may have an important and enduring effect on the success in life of such children and that early indicators of lead burden and behavioral deficit are strong predictors of poor school outcome. For the small group of 10 subjects who were diagnosed earlier as having plumbism, the outcome was especially dire; half of these young people have reading disabilities, and almost half left high school before graduation. Given the federal estimates that 16 percent of children in the United States have elevated blood lead levels (>0.7 μmol per liter [15 μg per deciliter]), the implications of these findings for attempts to prevent school failure are intriguing. The practical importance of early detection and abatement of lead in the environment, before it enters the bodies of children, is borne out by these long-term findings in young adults.

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CHILD LABOR IN 1990: Prevalence and Health Hazards

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INTRODUCTION

Child labor is defined as the paid employment of children less than 16 years of age. It is a common phenomenon in American society, and in the past five years the numbers of employed children have been increasing steadily. According to provisional data from the US Department of Labor, more than 4 million children in the United States were employed in 1988 (13, 26, 28). Legally employed child workers include the urban high school student working in a fast food establishment, the suburban 11-year-old delivering newspapers, and the rural child working on a neighbor's farm. Illegal child labor is also widespread. Despite the popular belief that this problem was remedied long ago, illegal child labor has persisted in the United States and appears, in fact, to be on the rise (41). Four-year-olds "help out" in factory sweatshops passing fabric between their mothers' sewing machines to increase the speed of piece work, while 14-year-olds work on legally prohibited machinery in belt and garment factories, bakeries, and butcher shops. Children do industrial homework on school nights, and they pick vegetables in fields still wet with pesticides.

In this review we discuss the benefits and risks of child labor, review its historical background, and summarize briefly the legislation governing child labor. We then discuss the current resurgence of child labor in the United States and consider the public health significance of work as a cause of injury in childhood.

BENEFITS AND RISKS OF CHILD LABOR

Employment offers numerous advantages to children, both in their own eyes and in the opinion of their parents, employers, and vocational counselors (27). Development of a sense of responsibility, discipline, and teamwork are frequently cited as benefits of work, and serve as bridges between the worlds of childhood and adulthood. Potential exists through work for development of new skills and improved organization. Also, the exposure of children to a variety of jobs and professions may influence in a positive way their future occupational choices. A good employer provides a role model and may evolve into a mentor. Work has historically offered youth economic opportunity, and it provides money that can be used for college savings, travel, to help feed the family, or for personal extras not otherwise available. As the number of children living below the poverty line has increased in the United States, and as available college financial assistance for those in the middle class has dwindled, these economic opportunities have in recent years assumed great importance. Finally, work offers youth a sense of personal worth for a job well done.

There are, however, potential disadvantages of child labor. These fall into two categories: (*a*) threats to education and development and (*b*) risks of injury, illness, and toxic exposure.

One of the principal hazards of child labor is interference with school performance. Employed children risk having inadequate time for school homework and increased fatigue on school days; teachers of children in areas where preholiday employment is common or industrial homework is escalating have noted declines in the academic performances of previously adequate students. These children are described as falling asleep at their desks, and they are unable to learn (27; Schiffley Embroidery cases, D. Come, personal communication). Even if they maintain their academic performance, working children are able to participate less actively in afterschool activities and sports than their peers. Child labor also interferes with play. Play is very important for normal development throughout childhood (30), and relaxation and freedom from fatigue are necessary for children to grow and learn. There is concern also that the push of youngsters into excessive job responsibilities may produce the "hurried child" effect described by Elkind (17).

Child labor may have further adverse effects on childhood development by encouraging antisocial behavior and by interfering with the formation of moral judgement (11). For example, the employer as role model may become a source of concern if his or her values, morals, and work habits are not those one would wish to inculcate in neophyte workers. When children are hired without working papers and asked to "work off the books," made to work after midnight, or asked to work on legally prohibited machinery, they receive socially inappropriate messages about the relative importance of the rule of law.

Child labor can also influence risk-taking behavior. Risk-taking is, to some extent, a normal part of adolescent development related to a lack of perspective on time (30) and a sense of immortality. If an adventurous adolescent is encouraged by unscrupulous employers to climb unprotected on scaffolding or to operate slicing equipment with the blade guard by-passed, however, he or she may develop cavalier attitudes toward risk-taking behavior that both increase inherent risk and lead later to injury and disability.

Injuries are the leading cause of death in children older than one year, and they account for 45% of all mortality in 5 to 14 year old children in the United States. Approximately 10,000 children die from injuries each year (44). Additionally, injuries are the leading cause of potential years of life lost (YPLL) in the United States, accounting for 2.3 million YPLL in 1987 (9). The risks of injury, illness, and toxic exposure associated with child labor appear to pose a significant public health problem, but they have only begun to be explored (36). Almost no data are available to characterize the rates of work-related injury among children. Major publications (6, 8, 18, 20) discussing the epidemiology of childhood injury fail to consider work as a causal or contributing factor. A recent review, however, of adolescent visits to emergency rooms in Massachusetts for treatment of injuries found that, of those injuries with an identified location, 24% had occurred on the job (2). In 1986, Workers Compensation awards were made to 1333 children under age 18 in New York State for work-related injury; 42% of these injuries involved some degree of permanent disability; 99 of the awards were made to children under the age of 15 (29).

Even less is known about the incidence or severity of work-related illness in children, even though children are known to experience a variety of toxic exposures at work. These exposures include formaldehyde and dyes in the garment industry, solvents in paint shops, organophosphate pesticides in agriculture and lawn care, asbestos in building abatement, and benzene in pumping unleaded gasoline. Given the occurrence of these exposures, it is not inconceivable that some still undefined fraction of adolescent asthma might be related to occupational exposures to dusts or formaldehyde or that some cases of adolescent leukemia may be the consequence of occupational exposure to the benzene in unleaded gasoline (33). Although it is recognized that young workers are exposed occupationally to substances known to be hazardous to adults, almost no work has been done to explore the possibility that young workers may have heightened susceptibility to these agents due to metabolic differences and increased body surface area compared to adults. Nor have possible risks in regard to causation of diseases with long latency been explored—a matter of concern, given that young workers have many more years of potential exposure.

In summary, although data are incomplete and studies preliminary, the available evidence suggests that work-related injury is a significant public health problem in children and suggests additionally that illness related to toxic exposure may be a problem in working children. Yet, children across the United States work daily with dangerous machinery, receive less than minimum wage, work overtime, and incur potentially hazardous occupational exposures.

HISTORICAL BACKGROUND OF CHILD LABOR

Child labor has a long history. In the Middle Ages, children worked in agriculture and as apprentices to artisans (19). In Colonial America, children who helped out on their own farms and households were commonly hired out to perform similar tasks at neighboring farms and households (a practice that has continued in rural areas almost without change). The contractual relationship that existed between employer and child was one that was supposed to recognize the responsibility of each to the other; although this did not (and does not) always preclude exploitation, proximity and the social interrelationships provided some degree of protection (31).

No such protection existed for children of the same era who were brought to the Colonies as indentured servants. Such children were often impoverished inhabitants of the streets of English cities. Gathered up by the hundreds and bonded for five to seven years of work in Virginia and other states, they were packed tightly into ships. Those who profited enormously from this system were not concerned about travel conditions; if the ocean crossing took too long due to bad weather, the food ran out. On one such trip 32 children died of hunger and disease and were thrown overboard. Of those who survived to reach America, many more died of disease within a few months (48).

Child labor underwent major expansion and restructuring during the 1700s as a consequence of the need, created by the industrial revolution, for large numbers of workers. In that era, "most mill owners preferred to hire children rather than adults. Above all, they were cheaper . . . but also more tractable, and as labor unions developed, less likely to strike" (39). Children as young as 11, especially girls, were sent by their families to work in the mills because wages they could earn far exceeded the income of their parents at home on rural farms. Not surprisingly, these young girls were often victims of sexual exploitation outside of the workplace in addition to exploitation inside the factories, where they commonly labored for 12 or more hours a day, six days a week (35).

The hazards and horrors of child labor in the eighteenth and nineteenth centuries were chronicled by Charles Dickens in *Hard Times* (14) and by Francis Trollope in *The Life and Times of Michael Armstrong, the Factory*

Boy (40). "Six-year old girls in the mines [of Scotland] did work that later, in times of relative enlightenment, was turned over to ponies" (24). In the mines of Pennsylvania and the mills of Massachusetts and South Carolina, conditions were no different.

> In the spring of 1903, [in] Kensington, Pennsylvania . . . seventy-five thousand textile workers were on strike. Of this number at least ten thousand were little children. The workers were striking for more pay and shorter hours. Every day little children came into Union Headquarters, some with their hands off, some with the thumb missing, some with their fingers off at the knuckle. They were stooped little things, round shouldered and skinny (48).

Drawings of children being beaten in the cotton mills, lowered on ropes into the coal mines, and carrying 50-pound rocks on their backs up mine ladders sparked great popular revulsion against the worst abuses of child labor, but still these continued (19).

In Britain, concern over the plight of working children was the principal stimulus to passage of the first legislation protecting the health of all workers (19, 39). The Health and Morals of Apprentices Act, passed by Parliament in 1802, fixed the maximum number of hours of work at 12 for apprentices, forbade night work, ordered the walls of factories to be washed twice each year and workrooms to be ventilated. In the United States, concerns about working children led to the enactment of compulsory education laws in the eighteenth and nineteenth centuries. For example, an 1874 New York State law mandated schooling for all 8 to 14 year old children and proscribed work on school days (39).

Despite federal and state legislation, child labor continued to be a major problem during the first third of the twentieth century. Inadequate enforcement of existing statutes contributed to this persistence. The need for enforcement was tragically demonstrated by the Triangle Shirtwaist Fire in New York City in 1911. Late in the afternoon one Saturday in March, a fire broke out on the eighth floor of a building that housed the Triangle Shirtwaist Company. Having recently lost their strike for a 40 hour week, 500 women and children on the seventh, eighth, and ninth floors of the building were still laboring, as part of their 59 hour work week. Since "the factory doors were locked each day to keep the (workers) in and the union organizers out" and the interior doors opened inward, fire escapes seemed the only recourse, but they broke under the weight of the desperate workers. The fire-engine ladders reached only to the sixth floor. Ultimately, 146 young women lost their lives that day, only eight years after the passage of landmark child labor legislation and fire protection laws in New York. Many of those killed were the sole providers for their widowed mothers and siblings (45).

Between 1916 and 1930, three major pieces of child labor legislation were enacted but struck down in the courts. As Postol recounts:

> In 1916 the first national child labor law, the Keating-Owen Act, was signed by President Wilson. This act prohibited the interstate commerce of goods produced by children under fourteen and established an eight-hour day for youngsters under sixteen. Just nine months after it was put into place, the Supreme Court ruled that Keating-Owen exceeded the federal government's power to regulate interstate trade, and the act was found unconstitutional. A second federal child labor law was enacted the following year, with the support of a potent reform group, the National Child Labor Committee (NCLC). It imposed a 10 percent tax on the net profits of manufacturers who employed children below the age of fourteen. In 1922, the Supreme Court struck down this act as an infringement on the rights of individual states to impose taxation measures. Having suffered two serious defeats, reformers became convinced that the only way to control child labor was through the passage of a constitutional amendment. Throughout the 1920s, the NCLC unsuccessfully sought to gain approval of the required number of state legislatures. Advocates of child labor reform were encouraged when, in the early 1930s, the National Recovery Administration banned child labor below the age of sixteen in most industries. In an all too familiar scenario, however, the NRA was invalidated by the Supreme Court in 1935 (31).

Finally, in 1938 the Fair Labor Standards Act was enacted. It remains the major federal legislation governing child labor today. This legislation established uniform federal standards for minimum wage, overtime pay, and maintenance of records on wages and hours for employees of all ages. Additionally, it established child labor standards, including lists of permitted work hours and prohibited occupations, and it raised the age limit for full-time work to 16.

Major reductions in child labor characterized the 40 years following passage of the Fair Labor Standards Act (FLSA). Although the tenets of the act helped to produce this decrease, Postol suggests that "a key reason the FLSA was effective was that child labor was already in decline by the time the bill was passed. By 1940, automation and structural shifts within the maturing American industrial economy had made child labor increasingly unprofitable. Changes in family size and demographics and restrictive immigration policies also contributed to the declining use of juvenile employment" (31). Widespread emphasis on the personal and societal value of education and a generally strong economy all combined to decrease the prevalence of child labor in most sectors. The major exception was in agriculture. Agricultural employment was exempted from many of the provisions of the Fair Labor Standards Act. Thus, the employment of children in agriculture remained common and is to the present time relatively under-regulated.

THE FAIR LABOR STANDARDS ACT AND THE WORK PERMIT SYSTEM

Under the Fair Labor Standards Act, no child under the age of 16 years may work during school hours, and a ceiling is set on the number of hours of employment permissible for each school day and each school week. Employment in any hazardous nonagricultural occupation is prohibited for anyone less than 18 years old. Thus, no one under age 18 may work in mining, logging, brick and tile manufacture, roofing or excavating, as a helper on a vehicle or on power-driven machinery. Meat processing machinery, delicatessen slicers, and supermarket box-crushers are specifically prohibited. In agriculture, where the restrictions are much less stringent, hazardous work is prohibited only until age 16, and all work on family farms is totally exempted. According to the law, however, no child under age 16 working on a nonfamily farm is allowed to drive a tractor with an engine over 20 horsepower or to handle or apply Category I or II pesticides and herbicides (25).

Though the FLSA provides a broad framework for the regulation of child labor, most administration of the law occurs on a state level, largely through the work permit system. Work permits are issued to children by state and local school systems. The intent of placing this authority within the schools was to allow for discretion in the issuing of permits based upon a student's academic performance. Thus the schools are also vested with the authority to rescind permission to work. In reality, however, this intended safeguard has been overwhelmed by other more pressing responsibilities placed on schools, and is virtually never exercised. Another problem in the administration of the FLSA lies in the fact that in most states there is no central collection point for data on the number or types of work permits issued to working children or the industries in which they are employed.

CURRENT EXTENT OF CHILD LABOR IN THE UNITED STATES

In the past decade, after 35 years in which it was not a problem outside of agriculture, child labor—both legal and illegal—has become increasingly common in the United States. The US Department of Labor estimates that 4 million children are legally employed; an additional large, but poorly defined, population is employed illegally (12).

A reconvergence of economic and social factors similar to those which produced the major increases in child labor at the beginning of the industrial revolution has produced this growing prevalence of child labor. These factors are:

1. A strong and growing economy in the face of low unemployment. This economy has generated a need for workers, whose numbers are scarce, particularly in the Northeast. Moreover, projected population declines and worker shortages suggest that this impetus to employ children will intensify in the years ahead.
2. Unstable world conditions, particularly war and poverty in Central America, which have led increasing numbers of illegal immigrants to enter the

United States. These immigrants, particularly children without parents, are highly vulnerable to exploitation in the workplace because of their overwhelming need for income and their fear of discovery by immigration officials.

3. Poverty: Despite a relatively strong economy, more American children live below the poverty line today than 20 years ago. For the 20% of American children who live in conditions of poverty, financial need constitutes a compelling reason to seek employment.
4. Relaxation has occurred over the past eight years in enforcement of federal child labor law, including the provisions limiting the maximum permissible hours of work and the prohibitions against use of dangerous machinery. Repeal of the ban on industrial homework, which was created to protect working women and children from industrial exploitation in this piece-work industry, has further undermined the historic intent of child labor law (23).

The illegal employment of children occurs in all industrial sectors, and often exists under sweatshop conditions (5, 32). Any establishment that routinely and repeatedly violates wage, hour, and child labor laws as well as the laws protecting occupational safety and health is termed a sweatshop (41). Traditionally, these shops have been considered fringe establishments, such as those in the garment and meat-packing industries. Increasingly, however, restaurants and grocery stores, not typically considered to be sweatshops, are also fulfilling the definition. In an effort to quantify the current problem of illegal child labor in the absence of readily available national statistics, the General Accounting Office (GAO) in 1987 surveyed the directors of state labor departments. Based on this survey, the GAO found in New York City that half of the approximately 5000 restaurants met the criteria for sweatshops, and that about 25,000 workers were employed in such establishments (42). In New York City fast-food establishments and the garment industry have been cited repeatedly for failure to pay minimum and overtime wages (4). The problem is not confined to large urban areas; in 1987, several high school students employed by a chain restaurant in a small West Virginia town quit after having tried unsuccessfully to negotiate with the manager to stop keeping them past midnight on school nights. Interestingly, the management had been no more responsive to a similar request made by a group of these children's parents (personal communication from adolescent patient, West Virginia University Hospital).

Health and safety conditions in sweatshops are often very hazardous. For example, fire hazards are created by blocked exit doors, accumulations of combustible materials, and inadequate ventilation, and electrocution hazards result from overloaded electrical supplies, work stations located close to exposed wire, and bare fuse boxes (S. Pollack, personal observation on visits to garment industry sweatshops in New York City in conjunction with the New York State Department of Labor Apparel Industry Task Force, 1988–1989). It is ironic that these same conditions are known to have contributed to the deaths of 146 women and children in the Triangle Shirtwaist Fire. The large number of fire code violations being discovered today by the Garment Industry Task Force inspectors of the New York State Department of Labor (H. McDaid, personal communication) suggests that workers, including children, are at very high risk of dying of fire if these conditions are not immediately alleviated.

HAZARDS OF INJURY ASSOCIATED WITH CHILD LABOR

Urban Child Labor

There are no recent epidemiologic studies of the health hazards associated with child labor in cities. The available information comes therefore from case reports and from evaluation of the types of employment available to urban children.

Garment industry sweatshop work appears to be an increasingly common source of employment for urban children. As noted above, fire, electrocution, and injuries from machines are all known risks associated with this work. Solvent exposure in leather shops is also a hazard. One of the most notorious sweatshops in New York City is a poorly ventilated belt factory that has received repeated child labor citations, and yet continues to employ children under the age of 16, all of whom spend the day in an atmosphere that reeks of glues and solvents (S. Pollack, personal observation). The persistence of such an establishment attests to the inadequacy of current law.

Stocking shelves and working at the cash register of grocery stores is legal work for children aged 14 and up, and urban grocery stores rely heavily on a young workforce. Although there is no literature concerning repetitive motion injuries among child cashiers, such injuries, including carpal tunnel syndrome, are known to pose problems for adult cashiers. The usual configuration of stock in big-city markets, where space is at a premium and items are stacked to a much greater vertical height than in other locales, poses risks both of ladder falls and of injuries from falling objects. Lacerations may be caused by cardboard boxes and the knives used to open them. Although children are prohibited by law from operating the machines that are used to crush or bale these boxes, an 11-year old boy in the Bronx was killed in December 1988, when he became entangled in a box-crusher and was crushed to death.

Delicatessen and bakery slicers, although their use is legally prohibited for children under 18, have both been shown to be sources of serious injury. In the early 1980s a teenaged boy in New York City was brought to the emergency room with an amputated arm; he said he had been "helping out" in a butcher shop. A few months later, another teenaged boy was brought to the same emergency room with his arm amputated after having been in the same shop. He too, had just been "helping out" (16). Recently, a 17-year old girl in New York City amputated several fingers when a bakery dough slicer came down on her hand (37).

The fast food industry is among the fastest growing industries and is one of the largest employers of youth in the United States today. Minor lacerations and burns are common hazards in fast food establishments. There is also a risk of electrocution, although this risk may have been lessened by changes mandated subsequent to the death by electrocution in 1987 of a teenaged worker in a hamburger restaurant; the source of the boy's electrocution was a power outlet on a wet floor in an improperly grounded building (15). Workers in fast food restaurants may also have excessive microwave exposure, as heavy use of the microwave equipment tends to damage the seals of food ovens; also in an effort to hasten the efficiency of food delivery, safety power cut-offs on microwave cookers may be circumvented (C. Gilman, personal communication). The extent of such exposure has not been quantified, but the potential is of concern due to the possibility that exposure to microwaves in high doses may cause eye damage with subsequent cataract formation.

Suburban/Small Town Child Labor

Children in small towns have a wide variety of job opportunities: delivering newspapers, pizzas, and submarine sandwiches, caring for lawns, working in gas stations, working in restaurants and fast food establishments, working at sales jobs in retail stores, and stocking shelves and working the registers in supermarkets.

The hazards associated with these suburban jobs are diverse. Lawn care is associated with mower injuries, including amputation of fingers and toes as well as eye injuries caused by flying rocks propelled by mower blades. Exposures to pesticides and herbicides can also occur in this industry. Newspaper delivery is associated with motor vehicle injuries to children on bicycles and on foot. Because unleaded gasoline in the United States contains 4 to 5% benzene by weight, employment of teenagers in gasoline stations may be associated with airborne and dermal exposure to this carcinogenic solvent. Finally, the delivery of pizzas and other hot food items has proven to be extremely hazardous to working children. The rash promise made by a midwestern pizza company that all pizzas would be delivered within 30 minutes of the time of placing an order has been shown to encourage reckless, dangerous driving by young, often inexperienced motor vehicle operators. A total of 20 fatalities among either children working in pizza delivery or the persons with whom they collided have been documented within the past year to be associated with the ill-conceived delivery policies of this firm (22; J. Kinney, National Safe Workplace Institute, personal communication).

Rural Child Labor: Farm Hazards

The work available to rural children is primarily agricultural, and poses the same health hazards as those affecting adult agricultural workers, including lacerations, amputations, and crush injuries from farm machinery; blunt trauma from large animals; motor vehicle accidents involving farm vehicles on public roads; risk of suffocation on grain elevators and silos; and exposure to pesticides. Small physical size and inexperience may, however, superimpose an additional risk for young workers. Although the numbers of children working in this industry are not so large as in other sectors, the potential hazards (especially those involving machinery and large animals), coupled with the historical lack of regulation of agriculture, combine to create an important problem, particularly in large rural states. Agriculture has come to surpass mining as the most dangerous occupation, accounting in 1980 for 61 fatalities per 100,000 workers (46). Perhaps for this reason, much of the scanty literature available on work-related injury and illness in children focuses on agriculture.

Data on injury in adolescent with workers are provided by a 1985 paper by Rivera (35).

> Nearly 300 children and adolescents die each year from farm injuries, and 23,500 suffer nonfatal trauma. The fatality rate increases with age of the child, the rate for 15- to 19-year- old boys is double that of young children and 26- fold higher than for girls. More than half (52.5%) [of those injured] die without ever reaching a physician; an additional 19.1% die in transit to a hospital, and only 7.4% live long enough to receive inpatient care. The most common cause of fatal and nonfatal injury is farm machinery. Tractors accounted for one half of these machinery-related deaths, followed by farm wagons, combines, and forklifts.

The importance of tractors in work-related agricultural injuries to children was supported by a 1979 paper on farm tractor fatalities in which Karlson et al stated that 29% of Wisconsin farm work fatalities in 1971–1975 occurred to male farm residents under the age of 19 (21).

Cogbill, Busch, and Stiers (10), also of Wisconsin, echoed the concerns of Rivera over deaths of injured child farm workers that occurred before they could reach a hospital. In a 1985 paper, they reviewed the cases of 105 farm trauma patients, 19 years old or younger, admitted to their Level II trauma referral center. They found a bimodal distribution in age with peaks at ages 4 and 14 years. All 13–18 year olds were working at the time of their injury (G. Stiers, personal communication). They found that "specific injuries observed in these children were often predicted by the mechanism of injury. Tractor and wagon accidents resulted in multiple system trauma with frequent pelvic fractures, long-bone fractures, head injuries, and thoracoabdominal trauma. Hand and leg trauma predominated in farm machinery mishaps." Six of the teenagers were critically injured. The most severely injured child (aged 15) spent 55 days in hospital after a tractor roll-over left him with fractures of skull, rib, pelvis, femur, ulna, face, both scapulae, and a laceration of the urethra that required surgical repair.

A bimodal peak of childhood farm injuries, in which the adolescent peak is accounted for by working children, was also noted by Swanson et al in Minnesota (38). They reviewed 88 cases of rural injury in adolescents, of whom 29 (33%) were definitely working at the time of injury. (Another 20 may have been working.) They found that older children were involved in accidents with tractors more than twice as often as younger children; power take-offs (rotating drive shafts which transfer power from a tractor to a piece of attached farm machinery) were a second important cause of injury. They provide sobering data on the implications of acute injury: "Eleven children required multiple reconstructive surgical procedures, fifteen children had a body part amputated, four children [were left with] arm or leg length discrepancies, four have nerve deficits, nine have diminished function secondary to contractures or deformities, and one has urethral deformities and impotence."

Cogbill et al point out that federal and state laws require all 14 and 15 year olds hired as farm employees to have completed a safety education course prior to operating machinery, but they add that these regulations do not apply to children working on family farms (10). Nevertheless, this safety requirement is not always being met even on farms where it is required by law. In 1984, a 16 year old New York State boy died while working on a neighbor's farm when the machine he was unloading caught and pulled him in, leaving him torn and crushed. He had been working since age 11, but had no work permit and no tractor permit. In testimony, his father accurately noted another problem pertaining to farm vehicle safety: Although one must have both a license and an inspected and registered car to drive on public roads, a farm vehicle need not have a certificate of inspection and can be driven by anyone, including a child with no license (27).

A 1989 paper by Broste et al from the Marshfield Clinic describes hearing loss among high school farm students (7). The paper is of historic as well as medical note, as it represents perhaps the only report of occupational illness in adolescents. Over three years, audiometric exams were conducted on 872 vocational agriculture students. Students who were actively involved in farm work had increased prevalence of high frequency, early noise-induced hearing loss as compared to peers who were not actively involved in farm work. Broste suggested that education and provision of hearing protection equipment would be appropriate preventive measures, as students using hearing protection had a lower prevalence of hearing loss. They concluded:

With the economic pressures facing many farmers, exposure of children to noisy and hazardous machinery could be expected to increase. Adolescents and teen-aged children often fill roles previously held by hired help. As farmers and their spouses take jobs off the farm to supplement income, farm children take on increasing responsibility for farm operations, and may be at increasing risk of hearing loss and other occupational health hazards of farming.

INTERNATIONAL CHILD LABOR

Child labor is a major problem beyond the borders of the United States (1, 3, 43, 47). According to the International Labour Organization (ILO), at least 200 million children worldwide under the age of 14 are employed. In some countries, children constitute 15 to 25% of the total workforce. Children are employed as rug weavers in the Middle East, underground tin miners in South America, metal workers, fireworks makers, textile weavers, and glass blowers. Injuries, illness, and disability are common among working children worldwide.

Child labor is associated in virtually all countries—both industrialized and developing—with poverty, high unemployment, inadequate educational opportunities, and failure to enforce existing law and standards. Particularly severe abuses have been documented in so-called "free enterprise zones," industrial areas established in many countries in which relaxation has been permitted in the enforcement of labor and environmental laws.

OPTIONS FOR PREVENTION

A series of approaches exists to prevention of the illnesses and injuries associated with child labor. All of these approaches are based on the fundamental preventive concept that the incidence of the injuries and illnesses associated with child labor will be greatly reduced if the laws and regulations governing child labor are understood and strictly enforced and if illegal child labor is eradicated. Although work in childhood has undeniable benefits, it is clear that excessive and illegal child labor, sweatshop work, and industrial homework are dangerous for children and must be prevented.

Specific approaches to prevention are as follows:

EDUCATION Education is necessary to call attention to the hazards associated with child labor and to provide information concerning the relevant law. It must proceed on several levels. Children themselves must be taught about the hazards of work, and an effort made to temper their usual enthusiasm and lack of fear of industrial hazards. Teachers and school boards must be informed of the hazards of child labor—including the hazards to education—and they must be made aware of their duties and responsibilities under the work permit process. Physicians and other health providers—particularly emergency room staff—need to remember that work can be a cause of injury and illness in childhood; the importance of an occupational history cannot be overemphasized. Finally, the business community must be educated as to the hazards of child labor and reminded of their responsibilities under the law.

DATA COLLECTION One of the major impediments to defining and resolving the problem of child labor in the United States is a lack of detailed data on the size of the population of working children and/or the incidence of work-related injuries and illnesses. The Bureau of Labor Statistics of the US Department of Labor and state departments of labor need to develop mechanisms for more efficient collection of these data and for the better accessing of data sets that are potentially useful, but currently only minimally available, such as information on work permits issued by school boards.

REGULATION AND ENFORCEMENT Relaxation in the enforcement of federal regulations protecting child workers has undoubtedly contributed to the current resurgence of legal and illegal child labor in the United States. The number of inspectors has been reduced, the number of inspections is down, and an attitude of complacent non-enforcement is officially sanctioned and all too common. A particularly dangerous proposal is the US Department of Labor's announced intention to relax bans on industrial homework that currently are in force in the apparel industry. Such relaxation will inevitably allow increased child labor, and is not consistent with good public health. Clearly, strong enforcement of existing legislation and regulations is necessary to prevent abuses of the law and to protect the health of working children.

CONCLUSIONS

Child labor—both legal and illegal—has increased substantially in frequency in the United States over the past decade. Childhood employment has increased in all industrial sectors, and the most marked increases have occurred in the restaurant and garment industries. A resurgence of sweatshops, many of them employing child laborers, has been documented by state labor officials and by the US Government Accounting Office. There has occurred a resurgence of industrial homework, often involving children. Violations of federal child labor laws have more than doubled. Currently, 4 million children in the United States are estimated by the US Department of Labor to be legally employed, and an additional large, but inadequately documented, population of children are employed illegally. Worldwide, the International Labour Organization estimates that 200 million children under the age of 14 years are employed.

Child labor, particularly when it is illegal, excessive or occurs under sweatshop conditions, can interfere with the emotional and intellectual growth of children. Excessive child labor has been shown to create major impediments to successful school work.

Child labor appears to be a serious cause of injury in children. Although statistics are scant and adequate epidemiologic studies have not been undertaken, lacerations, amputations, crush injuries, and head trauma have all been reported in children at work. Fragmentary surveys of worker compensation records and hospital emergency room records indicate that the possibility of injury to working children is substantial. Further epidemiologic studies of work-related injury to children are needed to better document the extent and severity of the problem.

Working children are exposed to numerous toxic substances, including known carcinogens (benzene and asbestos) and neurotoxins (organophosphate pesticides and solvents). Although disease resulting specifically from toxic exposures in childhood has not been documented, it seems probable that delayed illness consequent to these exposures must occur. Again, epidemiologic evaluations are required.

Prevention of injury and illness in working children requires the following actions:

1. the development of better data to define the extent and severity of the problem of child labor and its associated injuries and illnesses so as to permit identification of particularly dangerous industries and occupations;
2. education of children, parents, teachers, physicians, and the business community about the hazards of child labor;
3. review of existing laws and regulations to determine whether there are areas that require strengthening or modification so as to be more appropriate to current conditions;
4. discontinuation of the poorly considered federal initiative to relax certain labor regulations that protect children at work, particularly the regulations limiting industrial homework; this relaxation will lead inevitably to increased child labor;
5. strict enforcement of existing law and regulations by state and federal agencies.

ACKNOWLEDGMENTS

The authors acknowledge the generous support of the William T. Grant Foundation and the National Institute for Occupational Safety and Health (Grant # OH-02717). Also we thank Karen Garibaldi, Katherine Gleaton, and Mary Landrigan for their assistance in preparation of the manuscript. The helpful comments of Rob McConnell, MD, Marilyn Schiller, PhD, and Dorianne Beyer, JD, were greatly appreciated.

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September 26, 1990

Mr. George Miller, Chairman
House Select Committee on Children,
  Youth, and Families
385 House Office Building Annex 2
Washington, DC 20515

RE: Testimony for Hearing Record on "Environmental Toxins and Children: Exploring the Risks"

Dear Mr. Miller:

I am submitting the following comments to the Select Committee's hearing record on behalf of the Farmworker Justice Fund, Inc. (FJF). FJF is a national, not-for-profit farmworker advocacy organization based in Washington, D.C. One of our principal concerns is the workplace health and safety of migrant farmworker adults and children. We were pleased that the Committee included farmworker testimony in the Oakland hearing, and that you also addressed the issue of pesticide exposure of migrant children at the Capitol Hill hearing.

There are four points I will cover in this testimony:

1. The extent of pesticide exposure to migrant farmworker children;

2. Pesticide poisoning among farmworker adults and children;

3. The importance of pesticide recordkeeping; and

4. Other toxic exposures migrant farmworker children face.

1. The Extent of Pesticide Exposure to Migrant Farmworker Children

Farmworker adults and children are on the front lines of exposure to toxic agricultural pesticides. About 25% of farm labor is performed by children in this country. Migrant farmworker children, who themselves work or who are brought to the fields because parents have no day care, are exposed to pesticide residues in the soil and on the foliage of fruit and vegetable crops. Farmworker families get sprayed by pesticide drift when neighboring fields are treated. Sometimes farmworkers are not notified which fields will be sprayed, and the very fields where they are working get sprayed. Poisonings result.

Sometimes unprotected farmworkers are sent into the fields too soon after pesticide application. Such a violation of the pesticide reentry time in November 1989 resulted in the poisoning of 85 farmworkers, including several teenagers and pregnant women. (See attachment 1, newspaper clippings about that mass poisoning.)

Farmworkers suffer higher levels of pesticide residues in the food they eat and the water they drink than does the average consumer. Farmworkers are exposed to pesticides when they eat fruits and vegetables from the fields and when, because their employers provide no wash water, they must eat their meals in the fields without being able to wash their contaminated hands.

Farmworkers improvise when they are not provided with cups for drinking water. They use the very crops they are harvesting--green peppers, hollowed-out cucumbers or apples. If produce is covered with pesticide residues, the workers drink these residues along with the water. Migrant farmworker families may have to use pesticide-contaminated sources of water, such as irrigation ditches, for bathing, drinking, and washing because there are no other alternatives.

Migrant farmworker housing is typically located adjacent to the fields. When fields are sprayed with pesticides, drift carries and the farmworker camps are also sprayed. Thus, even if a family has a member who can stay with infants and small children at the labor camp, there is still the risk of pesticide exposure and poisoning. Additionally, improper disposal of pesticide containers by pesticide applicators may mean that areas near migrant labor camps become dumping grounds and hazardous playgrounds for migrant children. An article from June 1990 in Michigan's Grand Rapids

Press noted such a problem in a farmworker labor camp. (See attachment 2, Michigan section of "A Continuing Harvest of Shame.")

Children are also exposed to pesticide residues from their parents' work clothing since farm employers do not provide changing areas and shower facilities for farmworkers to use before they return to their living quarters.

Cases of Pesticide Poisonings among Farmworker Children

Attachments 1-4 include newspaper accounts of farmworker pesticide poisonings, including a farmworker applicator's death in California in early 1990 from the insecticide parathion. Contrary to what Committee witness Chris Wilkinson stated at the September 13th hearing, people have died from pesticides. Even when farmworkers are not fatally injured from a poisoning, they may be hospitalized or suffer lingering problems such as headaches, fatigue, problems with vision or sleep, and muscle weakness for days, weeks, or months after the poisoning. These chronic problems affect their ability to work and earn a living. Then there are the many unanswered questions about the long-term health effects of low-levels of exposure of children to a variety of pesticides over a number of years.

The effects of pesticide poisoning on children may lead to problems with learning and impairment of cognitive processes. A comparison of poisoned subjects and unexposed controls by Dr. Eldon Savage and colleagues at Colorado State University showed that the poisoned cases scored significantly worse in tests of intellectual functioning, academic skills, abstraction and flexibility of thinking, and simple motor skills (speed and coordination). (See attachment 5, pp. 69-70.) As former OTA study director Dr. Mark Schaefer stated at the September 13th hearing, though, little neurotoxicological research is devoted to vulnerable segments of our population, including children.

During the first weekend of September 1990, a crew of 75-100 pear pickers in eastern Washington were sprayed by pesticide drift. According to George Finch, the coordinator of Centro Campesino, Granger, Washington (phone number: 509-854-2052), the farmworkers are experiencing a range of health problems, including inflamed eyes, headaches, and rashes on the arms, hands, and abdomen. The following Monday none of the sprayed workers was rehired by the employer because "they had been talking to people" about the incident.

Employer intimidation of workers is an important factor in considering how well migrant farmworkers can protect themselves and their children from unsafe living and working conditions. The Farmworker Justice Fund has received reports from around the country of employers firing farmworkers whom they suspected of reporting pesticide, housing, or field sanitation violations or workers who complained to the supervisor or who merely asked questions about unsafe conditions.

We recently learned of another farmworker death from pesticide poisoning, which occurred in Georgia. An 18-year-old male died from exposure to the highly toxic insecticide disulfoton. The worker's job was to harvest pecans, which is done by shaking the trees. The young man was barefoot, had no protective clothing, and had received no warnings or training about pesticide safety. Shaking the trees was a dusty job, and we speculate that the residues of the pesticide in the dust were still highly toxic because of the very dry and hot weather. We are waiting for more information about this case.

3. The Importance of Pesticide Recordkeeping

Although industry-employed scientists such as Chris Wilkinson speak of the need for more data to adequately assess the effects of pesticides, the chemical industry and agribusiness have consistently and strenuously opposed passage of comprehensive pesticide recordkeeping requirements and worker and community right-to-know laws. Farmworkers, rural communities, and consumers are in a catch-22 situation. Examples of ill effects of pesticides are dismissed as "anecdotal evidence" or "unscientific." Yet efforts to secure comprehensive, accessible pesticide usage data are thwarted at the state and federal levels by well-financed campaigns by chemical manufacturers and the American Farm Bureau Federation.

FJF commends you, Mr. Miller, for your leadership and commitment to correct this problem through a recordkeeping amendment to the 1990 Farm Bill. Acceptance of the House version of pesticide recordkeeping by the Conference Committee would provide for farmworker access to pesticide information and meaningful data collection and analysis at the national level.

4. Other Toxic Exposures Migrant Farmworker Children Face

Attachment 2 shows some of the unhealthy and dangerous housing conditions migrant farmworker families contend with as they harvest our nation's food. Substandard housing puts migrant farmworker children at risk of exposure to various hazardous substances besides pesticides. Farmworker infants and children may be exposed to peeling lead-based paint, exposed asbestos insulation, and poorly ventilated heating systems.

Conclusion

Migrant farmworker children are exposed to a variety of environmental toxins in the fields and in the labor camps. I have focused on their exposures to pesticides. The surest way to protect children is to eliminate their exposure to pesticides in the first place. Pesticide poisoning is a preventable illness just as is lead poisoning. Substandard working and living conditions must be corrected. Farmworker parents must be able to earn a living wage without having to recruit their children to work so that the family can eat each day. Adequate child care facilities are needed for the children of farmworker and other rural parents as well as for the children of urban working parents.

Chris Wilkinson stated that "We must not squander our precious resources." He was talking about money. But children are our most precious resource, and we must protect them from environmental and occupational toxins.

We would be happy to provide the Committee with any additional information necessary. I have also attached a copy of our latest newsletter, a special report on pesticides.

Sincerely,

Valerie A. Wilk

Valerie A. Wilk, M.S.
Health Specialist

Attachments 1-6

ALL ATTACHMENTS ARE RETAINED IN COMMITTEE FILES

Attachment 1: "Deadly Insecticide Injures Farmworkers", article, from the Tampa Tribune, November 16, 1989.

Attachment 2: "A Continuing Harvest of Shame", Conditions Facing Migrant Farmworkers in 1990. The Farmworker Justice Fund, Inc., Washington, DC.

Attachment 3: "Pesticide Fatality a Mystery", article from The Bakersfield Californian, January 23, 1990.

Attachment 4: "Good Health Is a Mirage to Migrant Workers", article from The Grand Rapids Press, June 17, 1990.

Attachment 5: "The Occupational Health of Migrant and Seasonal Farmworkers In The United States", pamphlet from Farmworker Justice Fund, Inc.

Attachment 6: "Special Report: A Farmworker Perspective On Pesticides", article from Farmworker Justice News, Vol. 4, No. 2, Farmworker Justice Fund, Summer 1990.

Prepared Statement March of Dimes

STATEMENT ON ENVIRONMENTAL HAZARDS TO PREGNANCY

SUBMITTED TO THE
HOUSE SELECT COMMITTEE ON CHILDREN, YOUTH AND FAMILIES
SEPTEMBER 1990

The March of Dimes Birth Defects Foundation commends Chairman Miller and the Select Committee for holding these hearings on environmental hazards to children. The March of Dimes is concerned about the environment where children are first exposed to potential environmental risks -- the mother's womb. The risks to the fetus exposed to tobacco, alcohol, cocaine and other illicit drugs and some industrial byproducts (e.g. methylmercury, lead) are well documented. However, the risks to the fetus from exposure to many other drugs, industrial products, air pollution and water contamination are poorly understood.

Every year about 110,000 American children will be born with a serious birth defect (structural congenital anomaly or chromosomal abnormality). About 11,000 (ten percent) of these children will die and many of the others will have chronic disabilities. Birth defects have no class or racial boundaries, so that families from all socioeconomic groups share the same risk of having a child with a birth defect.

Birth defects are the leading cause of infant mortality in the United States. For those children who don't die, medical and institutional care costs billions of dollars annually, not to mention the cost in human suffering and anguish for the child and for the parents. Unfortunately, the causes of 80% of birth defects are unknown. Public concern about the safety of food, air, water and occupational exposure is rapidly increasing. Now is the time to address and answer some of these concerns, and we are pleased to have the opportunity to submit this statement to the select committee.

Our statement focuses on two opportunities for preventing environmental injury and damage to developing babies. The first is preconceptional counseling and prenatal care. Many of the medical conditions, personal behaviors, and environmental hazards associated with negative pregnancy outcomes can be identified and modified or treated prior to conception. Given the importance of minimizing the effects of environmental toxins on pregnancy outcomes, we should take advantage of every opportunity available to evaluate and counsel women of childbearing age who are considering a pregnancy, or are pregnant, about the risk of exposure to environmental toxins. Such encounters, if properly utilized, create an excellent opportunity for appropriate decision making that can provide the best chance for a good pregnancy outcome.

Pregnancy is often the impetus for a woman to seek health care following a period of either no care or episodic care. Unfortunately, access to early and continuous prenatal care is too often unavailable because of financial or other barriers. Any responsible solution to the problem of environmental hazards and children must address the issue of access to comprehensive primary health care.

The second opportunity to reduce environmental hazards relates to birth defects monitoring and research opportunities for developing effective prevention strategies. Since animal studies are poor predictors of risk in humans, epidemiologic research should be an urgent priority. Currently, the budget for these activities at the Centers for Disease Control (CDC) is less than 2% of the total CDC buuget. In fact, the state of California spends more than does the CDC for birth defects monitoring. California has the largest population-based birth defects registry, the California Birth Defects Monitoring Program (CBDMP). This program, funded by the state Department of Health Services and operated by the March of Dimes, is legislatively mandated to: maintain a birth defects registry; investigate clusters of birth defects; conduct epidemiologic studies about the causes of birth defects; evaluate environmental agents and provide community services directed at identified problems.

The CBDMP serves as an important epidemiologic resource to help identify causes of birth defects and to address public concerns about suspected causes and clusters. The CBDMP makes hundreds of public presentations annually and responds to hundreds of requests for information. The program has published studies on special environmental concerns. For example, one showed that aerial application of malathion was not associated with birth defects and another found that gastroschisis (a defect in which intestines are outside the abdominal cavity) is increasing over time among young mothers throughout California. The reasons for this increase are currently under study. One staff person from the CBDMP was instrumental in identifying accutane (an anti-acne drug) as a human teratogen.

While we are making great progress in California, this program does not substitute for a national program. The data from the CBDMP can be used nationally, however the California program does not have adequate funding for research activities. In addition, while other states also collect data, it is not collected in a uniform manner, which reduces the effectiveness of regional comparisons and our understanding of the epidemiological issues involved.

Therefore, the March of Dimes supports the expansion of the Centers for Disease Control to enable it to:

- stimulate and coordinate state efforts to monitor birth defects;
- help in planning further epidemiologic studies to identify causes of birth defects;
- fund epidemiologic research using existing data sources; and
- develop more effective prevention and intervention strategies based on the monitoring efforts.

We have much, much more to learn, not only about birth defects, but about the dangers from new environmental agents, chemical exposures, and illegal substances, not to mention a further evaluation of the 66,000 chemicals currently used in United States homes, industry, and agriculture. The March of Dimes, through its Reproductive Hazards in the Workplace, Home, Community, and Environment Program, will continue to support research in these areas; however, March of Dimes resources and even state resources are not enough. There is an important federal responsibility. We hope that these hearings will result in support for expanding the CDC birth defects prevention program to improve pregnancy outcomes for all American women.

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National Network To Prevent Birth Defects

Box 15309, Southeast Station, Washington, D.C. 20003, 202 543-5450

September 18, 1990

Hon. George Miller
Chairman
House Select Committee on
Children, Youth and Families
House of Representatives
Washington, D.C. 20510

Dear Rep. Miller and Collegues,

We appreciate that you are holding a hearing on "Environmental Toxins & Children: Exploring the Risks".

Enclosed is the testimony of the National Network to Prevent Birth Defects.

In our review of the literature over the past five years, it has become very clear that very large percentage reductions in birth defects, learning disability, mental retardation, and childhood cancers are possible, through a combined program of toxic exposure reduction and upgrading of nutrition during early pregnancy.

The federal government has goals for cancer and heart disease of adults, but lacks goals for improving the health and quality of life of children.

The Congress needs to lay out some goals for the regulatory agencies and departments.

We doubt very much that progress will be made in this area unless the Congress takes such action and promotes a presumption in favor of better health for children and the pregnancy in the regulatory process for toxics, radiation, and pharmaceuticals. The regulatory agencies are at present resistant to this idea that children need to be protected.

With best regards,

Erik Jansson

Erik Jansson, Nat. Coord.

c. Committee

PREPARED STATEMENT OF ERIK JANSSON, WASHINGTON, DC

The National Network to Prevent Birth Defects is a membership group founded in 1984, to fill a gap in citizen group and government efforts. At that time, there was no group that concerned itself with the effect of the entire spectrum of toxics, radiation, and drugs upon the pregnancy and the health of children.

New Interest In Childhood Health And Toxics

In 1984, there was very little interest in childhood health in the federal agencies. This is beginning to change for some of the following reasons:

1. The reduction of lead in gasoline produced a dramatic decline in the blood levels of Americans, and some indications of a reduction of learning disabilities in children, as well as strong expectations of a reduction of heart disease and cancer in American adults, and a likely reduction of birth defect rates.

2. In California, a series of laws has pushed the issue of prevention forward:

   a. The Birth Defect Prevention Act forbids the sale of pesticides in California that cause birth defects.

   b. Proposition 65 was passed by the voters, requiring the labeling of chemicals that cause either cancer or birth defects.

   c. The "Big Green" ballot initiative of California to be voted upon in November goes farther, forbidding the sale of pesticides that cause "reproductive injury". That is a term that is broad enough to include many childhood diseases including learning disabilities.

3. We also note some changed attitudes in the Bush administration that was missing previously. In May, Secretary Sullivan of the Department of Health and Human Services announced the formation of a Maternal and Child Health Bureau. And in September, the Department announced the opening of a Department for Womens' Health Research.

These are timid steps, because the Bureau does not have line authority and a research program just postpones action on measures that can be taken right now to sharply reduce childhood illness rates and improve pregnancy outcomes. But they are steps, nevertheless.

We recently wrote Administrator Reilly of the Environmental Protection Agnecy about establishment of a childrens' policy for toxics and pesticides, as well as radiation, and will have some meetings at EPA on this issue.

But again they want to push the issue down to the research level, which postpones obvious and inexpensive measures that can be taken right now.

LACK OF GOALS AND POLICY FOR CHILDHOOD HEALTH

The Department of Health and Human Services has goals for reducing rates of adult cancer and heart disease. But, there are no goals for reducing rates of birth defects, mental retardation, learning disabilities, and childhood cancers.

The Environmental Protection Agency lacks even a general policy on children, though it has long been known that toxic and radiation exposures particularly affect children and the pregnancy at dosages lower than for adults.

The time has come for the Congress to establish a general children's policy for the regulatory agencies, and a general presumption in favor of childhood health and for healthy pregnancy outcomes. This would have to cover toxics, pesticides, radiation, and also doctor prescribed drugs, doctor prescribed radiation, and food additives. (Radiation includes non-ionizing radiation.)

There is a need to require the regulatory agencies to set some goals to achieve better health for the child and pregnancy outcomes, and report back to the Congress on these programs.

And finally, there is a need for the Congress to set some goals for proper nutrition during pregnancy, since we found in our review of the literature that the interaction of toxics and nutrition is what determines whether there will be birth defects, mental retardation, or childhood cancer.

In short, what is needed is a coordinated program rather than the piecemeal programs of today that are fought out toxin by toxin. It is not possible to have a coordinated program without goals. Coordinated programs are always less expensive.

Exhaustion of Administrative Remedies

As an organization, we have exhausted our administrative remedies since 1984, in a series of citizen petitions to the Environmental Protection Agency, the Department of Health and Human Services, and other Departments.

Chart 1 lists these petitions, which were largely rejected, though we did make some progress in moving the lead issue along.

These petitions covered each of the major toxin areas that affect children that are listed on the next page.

Chart 1

REJECTED PETITIONS, 1984-90
Exhaustion of Administrative Remedies: Status of Petitions Filed With Federal Agencies Since 1984 on Prevention of Childhood Injury

breast milk safety 1. Petition to Environmental Protection Agency for a program to prevent breast milk contamination with chemicals, March 1, 1985, 37 pages. Status: Largely rejected.

doctor prescribed drugs 2. Petition to Department of Health and Human Services on Therapeutic Drugs That Cause Birth Defects and Learning Disabiities Without Providing Health Benefits, August 1, 1985 46 pages, Status: Totally rejected

Resubmitted on April 30, 1990, Status: Totally rejected,

3. Petition to change the label of phenobarbital to prohibit administration to pregnant women and children under the age of 3, unless they have proven epilepsy. April 30, 1990 Status: Under consideration.

toxic metals 4. Petition to Federal Agencies, particularly the Environmental Protection Agency, for a comprehensive program to limit exposure to toxic metals, January 9, 1986, 103 pages, Status: Rejected, though it helped with the lead removal program from drinking water. The Congress forced E.P.A. to limit American use of organotins, when the Agency refused.

low-level radiation 5. Petition to Federal Agencies, particularly the Environmental Protection Agency and Department of Health and Human Services to reduce American exposure to low-level radiation by 50 percent. 115 pages, Status: Largely rejected

nitrates 6. Petition to Federal Agencies to reduce American exposure to nitrates and nitrites by 50 percent, August 1, 1988, 94 pages, Status: Environmental Protection Agency rejected petition for drinking water because it would require changes in agriculture, and USDA rejected petition.

doctor prescriptions 7. Petition to Department of Health and Human Services to stop Medicaid payments for unnecessary drug prescription, X-rays and cesarean sections during pregnancy, 4 pages, Status: Department said that they did not want to set quality standards for Medicaid and rejected petition.

low-level radiation 8. Petition to Environmental Protection Agency to change its assessment of the cancer potential of radiation in view of updated findings by scientists concerning Hiroshima victims, November 11, 1987, 3 pages, Status: Agency rejected petition.

aluminum 9. Petition to Environmental Protection Agency to eliminate use of aluminum sulphate in treating America's drinking water since it was causing Alzheimer's disease and learning disabilities in children. April 24, 1999 6 pages and copies of scientific studies, Status: Agency will make a decision in one to two years.

It is important to note at the outset that the largest contribution of toxins to ill health of children takes place during the pregnancy itself. This is where the bulk of the medical care needs originate, from the birth defects, retardation, learning disabilities and childhood cancers produced by exposure to the fetus during pregnancy.

Dr. Alice Stewart of Great Britain points out that most childhood cancers are actually contracted in the mother's womb, since there is no other way to explain the time lag involved in the childhood onset of these cancers.

And so protection of the child from toxic injury needs to include the pregnancy period. The goals set for prevention by the Congress needs to include the pregnancy.

The major categories of toxins that affect children include the following:

- toxic metals: including not only lead, but also cadmium, mecury, arsenic, aluminum, manganese, organotins. And there are others.
- low level radiation, including doctor prescribed X-rays, radon, radium.
- breast milk contaminants, including dioxin.
- therapeutic drugs that are prescribed unnecessarily to the pregnant woman or to the young child.
- pesticides and nitrates.
- solvents and chemicals.
- non-ionizing radiation.

The House Select Committee on Children, Youth, and Families has been in existence for a number of years. It is true that the Committee lacks legislative authority, but most members belong to other committees that can introduce legislation.

So much is known about how to prevent childhood toxic injuries.

Perhaps the time has come to stop debating childrens' health issues, and to establish a legislative framework to require the regulatory agencies and mainline departments to develop prevention goals and programs that encompass the toxins beyond the traditional alcohol, cigarette and social drug exposures.

Without legislative prodding from the Congress, prevention will be a very unlikely event. And the Congress needs to be very specific about goals, and about the range of toxins to be addressed by the regulatory agencies.

And finally, as we will see next, there is also a need to set some goals on nutrition during pregnancy, since it is the interaction of toxins with nutrition that determines the rate of many birth defects as well as learning disabilities and cancers.

WHAT CAN BE ACCOMPLISHED WITH A COORDINATED FEDERAL PREVENTION PROGRAM

We were quite surprised in our review of the literature about how much progress could be made in prevention of childhood illness stemming from toxics exposures.

As already noted, a successful prevention program needs to combine a reduction of toxic exposure to the pregnancy and young child with a nutrition program that includes supplemental vitamins for the pregnancy.

The reason for this is that many toxins damage the child by interfering with nutritional elements. A good example is spina bifida, one of the three largest causes of mental retardation in the United States. Alcoholism and Down's syndrome account for most of the rest.

Recent studies sponsored by the National Institute of Health and others show that the daily use of a multiple vitamin containing folic acid during the first six weeks of pregnancy can reduce rates of spina bifida as much as 75 percent. A drug known to cause spina bifida, valproic acid, also aggressively depletes folic acid in the body.

In summary, we believe that the following is possible with a combined program of toxic exposure reduction to the pregnancy and young child and better nutrition for the pregnancy:

- Retardation: A 50 percent reduction in national rates does appear quite feasible.
- Birth Defects: Available evidence suggests that 50 percent reductions in rates appear feasible. But, birth defect rates appear to be increasing in the United States, except for the neural tube defects which are prevented by better nutrition.
- Childhood Cancers: The majority of these are caused by background radiation like radon and radium - particularly when exposure takes place during pregnancy. For this reason, we believe that a 30 percent reduction in rates may be feasible. Elimination of X-ray exposures during pregnancy are feasible, but the Department of Health and Human Services has chosen not to pursue this - and need some prodding from the Congress.
- Learning Disabilities: We believe that a 50 percent reduction of rates may be feasible.

THE NEED TO CAST THE PREVENTION NET WIDER

Chart 2
50 Percent Reduction in Rates Seems Possible Though the Literature Is Spotty

AGENTS	Exposure Rates	Learning Disabilities	Retardation	Birth Defects	Childhood Cancer
1. TOXICS					
PCB's	100%	Possibly 10.5% in Michigan	Dioxins can produce neural tubes	?	1-2% of Adult cancers in Denmark PCB's and DDT - Effect on childhood cancer probably larger
Chlorinated and Brominated Pesticides and Chemicals	100%	Significant	?	Indications in humans	
Toxic Metals	100%	23% in Wyoming (5 metals) Lead and cadmium major factors	Significant	46% of birth defects in Boston facilitated by lead	Lead is a potent cancer promoter in Swiss human study
Radiation Ionizing	100%	Significant	7% or more	Best guess 20% Radium in drinking water major factor in New York	75 percent 29% of childhood cancers in S. Dakota linked to radium in drinking water
Non-ionizing		?	?	?	15% of childhood cancers
Workplace	?	?	?	9% in Norway - Few American surveys	Significant source of childhood cancers
Pesticides	100%	?	?	Home pesticides cause defects-New S. Wales	Home pesticides major leukemia cause
Nitrates Nitrites	100%	Indications	Causes neural tubes	6.7% of defects in rural America	Significant cancer cause
2. DRUGS AND SOCIAL DRUGS					
Therapeutic Drugs During Pregnancy	44.5% in Connecticut	Probably similar to birth defect rate	Aspirin, valium reduce IQ. Many others impact	13% in Connecticut	?
Cigarettes	27.5% during pregnancy (½ pack or more)	Weak agent	?	Weak agent but highly synergistic with Valium and likely some toxics	Significant
Alcohol	47% during pregnancy (any)	Significant	Fetal alcohol syndrome major source	Birth defects at high doses	?
3. NUTRITION					
	?	31% of rates in rural Maryland Nutrition moderates toxics impacts	75 percent reduction in spina bifida	Significant rate reductions	Immune system loss

Chart 2 summarizes some of the recent literature findings on the impact of various toxins upon rates of childhood injury. Chart 3 summarizes a recent study on folic acid supplements during the first six weeks of pregnancy.

In a longer summary that we are sending to each member of the Committee, a more detailed look at the toxins and nutrition are presented. There is reason to believe that the vitamin supplements are effective against a wide range of birth defects, beyond spina bifida.

WHY THE CONGRESS NEEDS TO ACT

In March 1989, we sent an open letter to President Bush to ask for a national childrens' policy, similar to the one of Japan which was adopted in 1951, but also include specific programs for toxics exposure reduction to the pregnancy and young child, and nutrition during the pregnancy.

It is difficult to see how good progress can be made without goals, presumption in favor of childhood health during regulatory decisions on toxins, radiation, and drugs, and a general policy.

With a more coordinated program, as already noted, 30 to 50 percent reductions in rates of birth defects, mental retardation, learning disabilities, and childhood cancers do appear quite feasible.

Despite a more favorable environment today, we have to report that the Department of Health and Human Services is studiously trying to avoid dealing with the unnecessary doctor prescribed drugs and radiation exposure during pregnancy. The Congress needs to prod this agency into cutting off Medicaid payments for medical procedures known to be dangerous and unnecessary.

The Environmental Protection Agency does not have a childrens' policy at this time, and have had to be pushed into programs such as lead reduction, and have studiously avoided relating other toxic exposures such as cadmium, arsenic, radon, or radium pollutants to children or pregnancy outcomes.

Without Congressional proding and a legislative establishment of some goals and policy, movement towards better health will come very slowly and programs adopted will be much more expensive to the taxpayer.

Likewise in nutrition, the Department of Health and Human Services has refused to endorse the use of prenatal vitamins during the first six weeks of pregnancy. Apparently, they are not interested in reducing rates of mental retardation in the nation. And the WIC program does not allow payment for prenatal vitamins. The Congress really needs to prod on this issue in addition.

VITAMINS – Strong Prevention

Chart 3

A study of 23,491 women undergoing prenatal screening around 16 weeks of gestation found that vitamin intake made a big difference in neural tube birth defect rates.

The rate of neural tube defects was 3.5 per 1000 among women who never used multivitamins before or after conception, or who had used mutivitamins only before conception.

In contrast, the prevalence of neural tube birth defects was 74 percent lower, at .9 per 1000, among women using folic acid containing multivitamins during the first six weeks of pregnancy.

Where the multivitamins did not contain folic acid or where the vitamin supplements were begun after 7 or more weeks, the prevalance was similar to nonusers. This pinpoints folic acid as a major factor in prevention.

Neural tube defects is one of the three largest causes of mental retardation in the United States. Drugs that destroy folic acid, like aminopterin, cause neural tube birth defects in laboratory animals.

The study demonstrates the need to begin multiple vitamins early, even before conception and carry it through the pregnancy.

Folic Acid In First Six Weeks Of Pregnancy Pinpointed As Prevention Of Neural Tube Birth Defects

Table 3.—Prevalence of Neural Tube Defects According to Intake of Folic Acid–Containing Multivitamins and Their Time of Use*

Prevalence ratio of 1.00 is normal

		Weeks 1-6†		Weeks 7+ Only‡	
	None	Folic Acid +	Folic Acid −	Folic Acid +	Folic Acid −
No. of cases	11	10	3	25	0
Total	3157	10713	926	7795	66
Prevalence per 1000	3.5	→ 0.9	3.2	3.2	...
Prevalence ratio estimate	1.00	0.27	0.93	0.92	...
95% confidence interval	...	0.12-0.59	0.26-3.3	0.45-1.87	...

*As reported in response to the question, "Did you take a multivitamin in the first 3 months of pregnancy?" If yes, "What week of pregnancy did you start the multivitamin?" (In practice based on date of last menstrual period).

†Among women who took multivitamins in weeks 1 to 6, folate content was not known in 38 instances.

‡Among women who took multivitamins in weeks 7+, folate content was not known in 22 instances.

AUSTRALIAN STUDY WITH SIMILAR RESULTS

Bower and Stanley compared the vitamin intake from food for 77 mothers whose babies had neural tube defects against that of two control groups. One with 77 mothers had birth defects other than neural tube defects, and the other with 154 mothers had no birth defects.

It was found that the risk of neural tube birth defects fell sharply as free folate increased in the diet.

Indeed, the risk was 63 percent less between the highest and lowest dietary free folate intake in comparison with the first control group, and 75 percent less in comparison with the second control group.

VITAMINS PREVENT CLEFT LIP DEFECTS

A study by Tolarova of Czechoslovakia in 1982 would suggest that cleft lip birth defect rates can also be reduced with vitamin supplementation. Studies we have reviewed in previous newsletters would lead one to conclude that this may be true of birth defects in general - that they are responsive to vitamin intake, but more study is needed.

This study is a prospective survey of women, mostly from Bohemia, who had one previous child with a cleft lip (with or without a cleft palate).

The study group was given a supplemental multivitamin which contains 2000 IU of vitamin A, 1 mg of vitamin B_1, 1 mg of vitamin

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B_2, 1 mg of vitamin B_6, 50 mg of vitamin C, 100 IU of vitamin D_3, 2 mg vitamin E, 10 mg nicotinamide, and 1 mg calcium panthothenate.

The 85 study women were recommended to take three of the tablets above along with 30 mg of folic acid per day for at least three months prior to conception, and to continue taking these tablets until at least the end of the first trimester of pregnancy.

As can be seen from the table, cleft lip rates were sharply reduced at 1.1 percent of births compared to 7.4 percent for the groups without the vitamins. Furthermore, there seems also to be a much lower rate of spontaneous abortion in the vitamin taking group, thought this was obviously not part of the study design.

UNILATERAL CLEFT LIP WITH OR WITHOUT CLEFT PALATE

	Outcome of pregnancy			
	Total	Spontaneous abortion	No cleft	Clefts
Supplemented	85 *(80)**	1†	83	1
Control	212 *(202)**	6‡	191§	15

*Figures in parentheses show no. of mothers.
†Examined, no cleft. ‡Not examined. §1 hypospadias, 1 with talipes equinovarus.

Source:
Mulinsky, Aubrey, et al, "Multivitamin/folic acid supplementation in early pregnancy reduces the prevalence of neural tube defects", JAMA, Vol. 262, Nov. 24, 1989
Bower, Carol and Fiona J. Stanley, "Dietary folate as a risk factor for neural-tube defects: evidence from..Western Australia", Med. J. of Australia, June 5, 1989, V. 150
Tolarova, M., "Periconceptional supplementation with vitamins and folic acid to prevent recurrence of cleft lip", The Lancet, July 24, 1982

APPENDIX

ADDITIONAL INFORMATION ON RISKS TO CHILDREN FROM TOXICS

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2. Background Radiation Like Radon Are Primary Source of Childhood Cancers. Radium Is Also A Primary Source of Birth Defects, When Found in Drinking Water. 6-8

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OTHER TOXIC METALS LIKE CADMIUM CAUSE LEARNING DISABILITIES

PART 2. THE OTHER TOXIC METALS AND LEARNING DISABILITIES

There is a Need for the Environmental Protection Agency to Develop a Prevention Program for Learning Disabilities Caused by Exposures to Metals

A series of recent studies indicate that lead is not the only toxic metal that causes learning disabilities. We have already seen Pihl and Parks summary of their findings about Canadian children (page 3) that five metals- cadmium, cobalt, manganese, chromium, and lithium predicted which children would be learning disabled with a 98 percent accuracy, a result that was totally unexpected.

The studies we are going to review indicate that five metals- lead, cadmium, mercury, arsenic, and aluminum can cause learning disabilities and can change behavior in children at dose levels that are often found among children in the United States. These findings thrown a new dimension on the debate about acid rain. Widespread and uncontrolled metal leaching can only produce adverse health effects of considerable cost and magnitude.

A comprehensive metals approach is needed by the Environmental Protection Agency. It should also be noted that since the controversy over the Needleman studies of Boston children a few years ago, a series of studies on the metal lead have confirmed his findings. Some confirming studies are described in appendix 1.

Study of Wyoming Children by Charles Moon et al

For example, Charles Moon et al's study of Wyoming children found that the combination of lead, arsenic, cadmium, mercury, and aluminum accounted for 23 percent of the variation of test scores for reading, spelling, and visual-motor performance in a group of Wyoming children. It seems likely that low dosage exposure to these metals may be more important than the teacher in explaining classroom results.

This study looks at metals as a whole, interacting with each other, rather than just one metal like lead. The metal levels are measured in the hair, rather than blood, which gives a longer sample of exposure.

Individual metals have different effects on the performance of the child, suggesting an affinity for different parts of the brain chemistry. The following results were found:

1. For spelling, arsenic accounted for almost 8 percent of the variation in test scores.
2. For reading, arsenic contributed 11 percent of the explained variation.
3. Lead and arsenic interacted to produce additional variation.
4. For visual motor performance, aluminum accounted for almost 9 percent of variation.
5. The interaction of lead and aluminum accounted for an additional 8 percent of the outcome variance.
6. And finally, all the metals together accounted for 23 percent of the test score variance in reading, spelling, and visual-motor performance.

Table 3
Subjects' Metal Concentrations

Metal	Mean	SD	Range	Accepted Upper Limit (ppm)*
Lead	6.50	2.86	2.00 - 15.00	15.0
Arsenic	2.88	1.27	0.30 - 5.88	7.0
Mercury	0.87	0.43	0.20 - 2.30	2.5
Cadmium	0.64	0.40	0.17 - 1.98	1.0
Aluminum	9.53	5.06	1.00 - 23.00	30.0

*Doctor's Data, Inc. (1982)

Moon (1985)

Moon et al's study show
significant learning
disabilities produced
toxic metal levels that
are below E.P.A.'s
assumed danger levels.

Lead and Cadmium in Rural Maryland Children and Learning Disabilities - Study by Thatcher et al

Thatcher found that the more rural children in Maryland often had higher lead levels, as measured in hair, than those of the regional towns. Lead in paint may account for some of this.

The 1983 study of Thatcher et al looks only at the metal lead, and its relationship to test scores for 149 rural children. Figure 1 is presented in the Executive Summary of this petition, and raises the question of whether a child can be gifted if exposed to anything more than low levels of lead.

It was found that lead accounted for 16.54 percent of the variation of test scores for the full scale IQ, 18.22 percent of the variation in performance IQ, and 9.62 percent of the variation of the verbal IQ.

There was no "threshold" below which these deleterious effects on the children's performance did not take place, which of course, is also the finding of Needleman using dental lead levels - appendix 1.

In 1982 and 1985, Thatcher and Lester reported on a two metal interaction study of 150 children. They compared the effects of lead and cadmium as measured in hair both with test scores, and with electroencephlograph (EEG) measurements of brain wave patterns.

It was found that both lead and cadmium were associated with reductions of neuronal excitability - basically reduced brain function in these children. As the concentrations of cadmium and lead increased, the response time or latency increased, and the brain waves showed smaller amplitudes. Furthermore, with higher concentrations of lead or cadmium, an increase in the amount of slow wave activity of the EEG was noted. (See page 16)

One of the most interesting findings was that lead and cadmium may affect different parts of the brain - a finding also suggested by Moon's study. For example:

1. Lead independently accounted for a significant amount of the performance but not the verbal IQ variance. See figure 5.
2. The reverse was true for cadmium - affecting verbal IQ significantly.
3. This was also shown in the analysis of the EEG findings.
4. It is believed that this may be due to the affinity of lead for the metabolism of the neurotransmitter dopamine, while cadmium's most direct effect is on norepinephrine, serotonin, and acetylcholine metabolism.

Figure 5

Prediction of WISC-R Scores by Cadmium after Regressing Out the Effects of Lead and the Prediction of WISC-R Scores by Lead after Regressing Out the Effects of Cadmium

	Cadmium Only (after regressing out the effects of lead)				Lead Only (after regressing out the effects of cadmium)			
	Partial R	Partial R^2	F	Power	Partial R	Partial R^2	F	Power
Verbal I.Q.	.1563	.0244	3.65*	.31	.0971	.0094	1.39	–
Performance I.Q.	.044	.0017	0.28	–	.2191	.048	7.361†	.81

*P < .01.
†P < .001.

Thatcher (1982)

Once again, there was no threshold below which either cadmium or lead did not produce adverse effects. Instead, the greater levels of either of these metals in the hair of the children, the lower the performance of the child in a continuum. (Fiqure 1- Executive Summary)

> "Analysis of the relationship between lead and cadmium indicated that there is a gradient effect with the higher levels of cognitive functioning affected at even low concentrations and gross motor movement affected at only higher concentrations...Furthermore, the fact that these negative effects were observed in a rural population of school children suggests that this phenomenon may be geographically pervasive." Thatcher (1985)

Study of Toxic Metals and Learning Disability in Canadian Children

It was found, using a discriminant function analysis, that the hair contamination levels of five metals permitted the prediction of which children could be classified as learning disabled with a 98 percent accuracy, a result that was totally unexpected. These metals were cadmium, cobalt, manganese, chromium, and lithium.

Lead was not included in this metals group because the metals cadmium and cobalt served in its place. There was a strong negative correlation between lead and cobalt levels in the hair (possibly because these metals complete with each other). There was a strong positive correlation between lead and cadmium. The more cadmium in the hair, the more lead was found also. (Note also the relatively elevated mercury content in the hair of these children, a sign that these Canadian families probably eat a moderate amount of fish.)

Pihl and Parks Study of Canadian Children

Five metals marked predicted learning disabled children with 98 percent accurancy, a result that was totally unexpected

Table 1. Mean element scores (expressed as mean parts per million (ppm)) for the two groups (LD children and controls) and analysis of variance (d.f. = 1, 47) for each element across groups (N.S., not significant).

Element	LD (ppm)	Control (ppm)	F ratio	Level of significance
Calcium	397	344	1.75	N.S.
Magnesium	35	37	0.28	N.S.
Potassium	1359	1240	0.21	N.S.
Sodium	1637	919	5.10	$P < .02$
• Cadmium	1.72	1.08	84.52	$P < .001$
• Cobalt	0.16	0.23	35.00	$P < .001$
Copper	12	17	0.72	N.S.
Iron	23	22	0.82	N.S.
Lead	23	4	28.32	$P < .001$
• Manganese	0.83	0.58	15.21	$P < .001$
Zinc	139	140	0.10	N.S.
• Chromium	0.25	0.09	8.49	$P < .01$
• Lithium	0.22	0.40	7.29	$P < .01$
Mercury	14	15	0.52	N.S.

Pihl and Parks, 1977

LEAD

LEAD AND PERMANENT MENTAL LOSS

Needleman and collegues recently followed up 132 young adults who had been studied as primary school children between 1975 and 1978.

The young people reexamined in 1988 were found to have lost mental capacity that was related to their lead exposures as measured by the lead content of their teeth shed at the ages of six and seven - about eleven years previously.

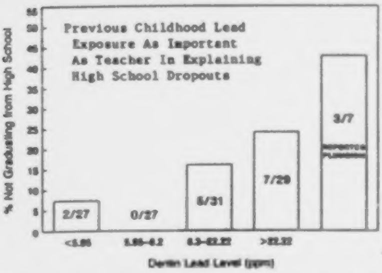

Figure 1. The Proportion of Subjects Who Did Not Graduate from High School, Classified According to Their Past Exposure to Lead.

For the young people with tooth lead levels in excess of 20 ppm eleven years previously, there was a markedly higher rate of dropping out of high school, (OR = 7.4), and having a reading deficit, (OR = 5.8), as compared to those whose tooth lead levels were less than 10 ppm in childhood. (OR = 1 is normal.)

Higher lead levels in childhood were also associated with lower class standing in high school, increased absenteeism, lower vocabulary and grammatical reasoning scores, poorer hand-eye coordination, longer reaction times, and slower finger tapping.

In summary, lead exposure during childhood produces permanent damage to the nervous system, and effects on school performance that are easily as large as the effects of the teachers.

Sources- Toxic Metals Other Than Lead

Thatcher, R.W. et al, "Nutrition, Environmental Toxins, and Computerized EEG...", J. of Learning Disabilities, May 1985, Vol. 18

Moon, Charles, et al, "Main and Interactive Effects of Metallic Pollutants..", J. of Learning Disabilities, April 1985, Vol. 18

Pihl, R.O. and M. Parkes, "Hair Element Content in Learning Disabled Children", Science, Vol. 198, 1977

Source:

Needleman, Herbert, L., "The long-term effects of exposure to low doses of lead in childhood", New England J. of Medicine, Vol. 322, No. 2, Jan. 11, 1990

Florini, Karen, L. et al, Legacy of Lead: America's continuing epidemic of childhood lead poisoning, Environmental Defense Fund, Washington, D.C., March 1990

RADON

78 Percent Correlation of Childhood Cancer With Radon

Radiation's health effects have been downplayed over the years to protect the defense industry and strangely enough, the medical profession. In February, 1990, for example, the Mayo Clinic Health Letter wrote that it was alright to have your fetus exposed to X-rays without shielding.

Chernobyl has changed a lot of minds about radiation, and has opened up the medical journals to articles that would have never been published ten years ago. These new studies come in as genetic damage is shown in plants and animals in the vicinity of Chernobyl and the evacuation zone is widened around the plant.

The Hazard of Radon Upgraded

Radon has been also downgraded as a health hazard by a long line of experts who have often consulted for the defense industry.

In our Spring 1989 newsletter, we reported on Kneale and Stewart's computer summary that background radiation in Great Britain accounts for about 75 percent of all childhood cancers.

A new study by Henshaw and collegues of the University of Bristol confirms such a high figure. In their study, there was a 78 percent correlation of childhood cancer against radon exposure over 12 nations and regions.

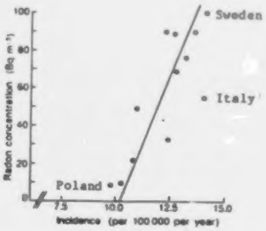

Fig 3—Radon concentration versus international incidence of all childhood cancer.

See table a for breakdown of cancer types. Radon values were taken as for country data in fig 1 and table a.

They conclude that for all age levels, 6-12 percent of all myeloid leukemias in the United Kingdom may be attributed to radon. In Cornwall, where radon levels are higher, the range is 23 to 43 percent. And they estimate that 13 to 25 percent of myeloid leukemias of all ages in the world may be caused by radon.

They estimate the bone marrow dose by assuming that the radon and breakdown radiation accumulates in the fat cell of the bone marrow, which is a new approach.

It appears that the time has come for the Environmental Protection Agency to renew and upgrade its program of reducing radon exposure to Americans in their homes and workplaces.

Source:

Henshaw, Denis, L. et al, "Radon as a causitive factor in induction of myeloid leukaemia and other cancers", The Lancet, April 28, 1990

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NEWS ON RADIATION 7

British Researchers Conclude That 75% of Childhood Cancers May Stem From Background Radiation

The largest registry of childhood cancer cases in the world is the Oxford Survey, put together by Dr. Alice Stewart and G.W. Kneale. Recently, the National Radiological Protection Board has put together a complete grid analysis of background radiation for Great Britain. And so, it became possible to compare childhood cancer rates with differing background radiation levels.

In 1986, Kneale and Stewart released a paper at the International Conference on Biological Effects of Ionizing Radiation in London, that studied the effects of two leaking nuclear reprocessing plants and background radiation upon rates of cancer in children aged 0 to 16.

As can be seen from the table, the study concludes that about 75 percent of all childhood cancer deaths may be caused by exposure to background radiation of various kinds. Previously, Stewart had also estimated that 6 to 7 percent of childhood cancer deaths are produced by unnecessary X-ray exposures during pregnancy.

An Explanation for Cancer Clusters

Experts for industries that use radiation often assert that cancer clusters are simply mathematical noise. Stewart and Kneale have a more convincing explanation based upon the concept that cancer is an immune system disease, and that children who do not die of cancer due to radiation exposure often die of infection instead.

In those areas where there is apparently low childhood cancer rates in spite of higher background radiation exposure, there is often an infectious disease incidence like measles to explain this. This balance between cancer and infection produces the apparent clustering of cancer cases by region.

Kneale, G.V. and A.M. Stewart, "Childhood Cancers in the U.K. and Their Relation to Background Radiation" for Proceedings of the International Conference on Biological Effect of Ionising Radiation, London, Nov. 24-5, 1986, c/o Regional Cancer Registry, Queen Eliz. Medical Center, Edgbaston, Birmingham, B15 2TH, Great Britain

Table 1
Radiation Contribution to Juvenile Cancers (0 to 16 years of age) in Great Britain

Radiation Source	Percent of Juvenile Cancers Produced
1. **Fetal Exposure to All Sources of Gamma Radiation**	At least 75 percent
2. **Fetal Exposure to Terrestrial Sources of Gamma Radiation (includes all uranium breakdown products such as radium in the drinking water, and radon) (20 to 27 percent total gamma dose)**	At least 15 to 20 percent
3. **Weapons Fallout (4 percent of gamma levels added)**	At least 3 percent
4. **Medical X-rays During Pregnancy**	6 to 7 percent

Gamma radiation from terrestrial sources, as compared to cosmic radiation, includes all uranium breakdown products, such as radium in the drinking water and radon. Gamma radiation itself has a significant cancer effect, but it could also in this study be

PANEL 3

Birth Defects and Background Radiation in New York State

In 1959, Gentry and collegues investigated the impact of natural radiation from various rock formations in New York State that contain uranium and thorium, on birth defect rates. Table 4 summarizes their findings.

Malformation rates per 1,000 live births in areas classified as having "probable" or "unlikely" presence of rock and soil with significant radioactive content: (New York State outside of New York City, 1948-55)

	Probable High Radiation Areas	Unlikely High Radiation Areas	Total
Total	15.1	12.8	13.2
Urban	14.1	12.8	12.9
Rural	15.8	12.9	13.4
Adirondack Highlands	17.8	12.1	
Catskill-Pocono Highlands	14.2	14.0	
Northern Allegheny Plateau	16.1	14.6	
Northwestern Allegheny Plateau Border	15.3	13.7	
Erie-Ontario-Mohawk Plain	14.8	12.1	
Hudson Valley and Long Island	15.4	13.3	

Radon measurements by the Radiation Branch of the New York Operations of the Atomic Energy Commission found that external radiation levels associated with the igneous rock formations ranged from .07 to .11 Rems/year, a range quite similar to Ujeno's study of Japan. The lowest background radiation level would correspond with rural birth defect rates of 12.9/1000, whereas the highest background radiation levels with rates of 17.5/1000 live births.

Also, water contamination with Radium was found, and produced the following birth defect rates:

Radium in the Drinking Water Appears To Be A Major Cause Of Birth Defects

Table 5

Source of Public Water Supply	"Probable" Areas			"Unlikely" Areas		
		Malformations			Malformations	
	Births	No.	Rate	Births	No.	Rate
Total	50,333	732	14.5	150,742	1,936	12.8
Wells and springs	15,307	259	16.9	60,994	783	12.9
Large surface (lakes, rivers)	11,196	139	12.4	31,275	371	11.9
Small surface (brooks, streams)	11,213	149	13.3	24,008	304	12.7
Mixed	12,618	185	14.7	34,545	478	13.8

Birth defects covered an entire spectrum.

Malformation Rates/ 1,000 Live Births

Table 6

Malformation Classification	"Probable" Areas		"Unlikely" Areas	
	Malformation	Rate	Malformation	Rate
All malformations	1,899	15.0	5,635	12.9
Central nervous system	254	2.2	857	2.0
Circulatory system	299	2.6	939	2.1
Hare lip, cleft palate	181	1.6	529	1.2
Digestive system	122	1.1	381	0.9
Genitourinary system	150	1.3	440	1.0
Skeletal system (club feet)	264	2.3	779	1.8
Bones and joints	140	1.3	466	1.1
Other unspecified malformations	356	3.1	1,101	2.5

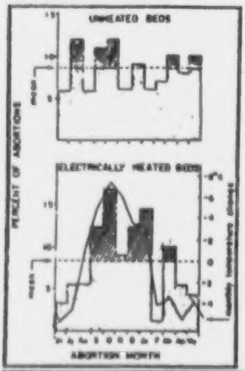
Source: Gentry, John, T. et al, Am. Journal of Public Health, Vol. 49, No. 4, April 1959

Non-ionizing Radiation

WATER BEDS AND ELECTRIC BLANKETS SHARPLY INCREASE MISCARRIAGE RATES

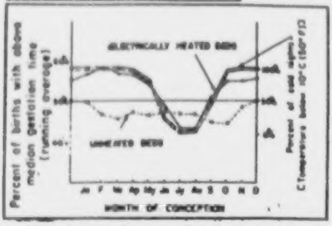
Nancy Wertheimer of the University of Colorado Medical Center and physicist, Ed Leeper recently reported on a study of 1700 Denver births over the 1976-1982 period.

It was found that the use of either water beds or electric blankets during pregnancy sharply increased the rate of miscarriage, as can be seen from the charts. The electric field of the water bed is the source of these miscarriages, and the field could be easily shielded notes Wertheimer. What to do about electric blankets is more difficult - best to discontinue use during pregnancy.



Increased Miscarriages (side)

1 Week Increase in Gestation Time (below)



This is the latest of a long series of reports linking ELF (extremely low-frequency magnetic fields) to cancer, suicides, abortions, and possibly birth defects.

Edwards, Diane, D., "ELF: The Current Controversy", Science News, Vol. 131, Feb. 14, 1987

10

NON-IONIZING RADIATION

There is growing evidence that magnetic fields created by electric wiring and equipment, such as word processors, can produce birth defects, miscarriages of pregnancy, and cancers. In the June - August 1987, issue, we reported on the sharp increase in miscarriage rates produced by sleeping on water beds during pregnancy. The magnetic fields dosage produced by water beds, of course, is quite high compared to many other appliances.

Likelihood (Adjusted Odds Ratio) of Miscarriage of Pregnancy and Birth Defects When Women Use Video Display Tube Word Processors During the First Trimester of Pregnancy, (Working Women)

Note: Odds Ratio of 1 is Normal

	Miscarriage		
	Number		
VDT	Miscarriages	Live births	Odds ratios
No VDT	240	310	1.0
<5 hr/wk	29	77	0.9
5–20 hr/wk	34	71	1.0
>20 hr/wk	52	65	1.8
Total	355	723	-

	Birth Defects		
	Number		
VDT	Birth defects	Live births	Odds ratios
No VDT	65	310	1.0
<5 hr/wk	8	77	0.9
5–20 hr/wk	12	71	1.4
>20 hr/wk	12	65	1.4
Total	97	723	-

ELECTRIC WIRES- Childhood Cancer in Denver - 15 Percent of all Cases?

David Savitz and collegues of the School of Public Health of the University of North Carolina, recently completed a study of 356 juveniles, aged 0 to 14, who had cancer - compared to an equal number of controls.

They found that exposures to magnetic fields from the electric wires delivering electricity to the house from the street, sharply increased cancer rates in children. These magnetic fields were measured by turning off all of the appliances within the house, to isolate the effect of the delivery wires.

As can be seen from the tables, measured magnetic fields under this lower power use had significant effects on childhood cancer rates. Wire codes associated with higher magnetic fields had a strong effect, varying with dosages across cancer subgroups, except for brain cancer and lymphomas.

WORD PROCESSORS- Kaiser Permanente Finds a Significant Increase in Miscarriage from Word Processors

The numerous complaints about miscarriage of pregnancy and birth defects associated with the use of video display word processors during pregnancy has spawned a number of studies.

Goldhaber and collegues recently reported on a case-control study of 1,583 pregnant women who attended three Kaiser Permanente obstetric and gynecology clinics in California. They found a significantly elevated risk of miscarriage for working women who reported using VDTs for more than 20 hours a weeks, during the first trimester of pregnancy.

Birth defect rates also rose 40 percent among moderate and high users, but the results were not statistically significant. The authors conclude that the possibility remains open that the VDTs themselves are hazardous to the pregnant operator.

Goldhaber, Marilyn, K. et al, "The Risk of Miscarriage and Birth Defects Among Women Who Use Visual Display Terminals During Pregnancy", Am. J. of Industrial Medicine, Vol. 13, pp. 695-706, 1988

ELECTRIC HEATING- Fetal Loss Due to Electric Home Heating

Wertheimer and Leeper in 1989 published a second study to follow their study on electric blankets or heated waterbeds and the effect on pregnancy outcomes.

Looking at rates of fetal loss in Oregon mothers living in homes with or without ceiling cable electric heat, which provides

It is estimated that 20 percent of the Denver area population is exposed to relatively elevated magnetic field levels from electric power distribution lines. The proportion would be lower in rural areas and higher in more densely populated areas.

An estimate that electric power distribution lines may account for as many as 10 to 15 percent of all childhood cancers nationally has been ventured.

Source: Savitz, David, A. et al, "Case-Control Study of Childhood Cancers and Exposure to 60-Hz Magnetic Fields", Am. Journal of Epidemiology, Vol. 128, No. 1, 1988

ome Electric Delivery Wires and Cancer Rates in Youth Aged 0 to 14

ancer Risk in Relation to Wire Configuration Codes: Denver, Colorado, SMSA
Note: Odds Ratio of 1 is Normal

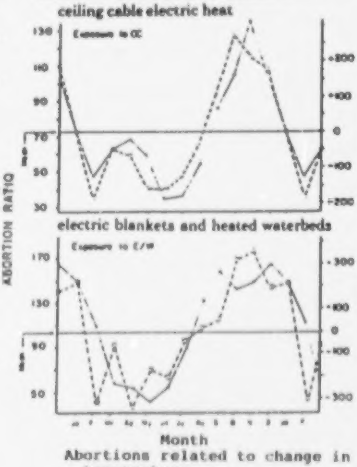
Wire code	No. of cancer cases (n = 356)	No. of controls (n = 278)	Odds ratio
Five-level wire code at time of diagnosis			
Buried	95	88	1.00
Very low	29	17	1.58
Low	107	102	0.97
High	70	44	1.47
Very high	19	8	2.20
Missing	36	19	
Mantel chi for trend = 2.03, p = 0.02			
ive-level wire code two years before diagnosis			
Buried	36	47	1.00
Very low	11	15	0.96
Low	50	56	1.17
High	30	28	1.40
Very high	8	2	5.22
Missing	221	130	
antel chi for trend = 2.31, p = 0.01			

a magnetic field ten times higher than the more commonly used baseboard heating, it was found that this electric heat increased the miscarriage rate much in the same way as had the electric blankets and waterbeds.

As can be seen from the figure, the relationship to cold weather and heating days was very similar, and also was statistically significant.

Wertheimer, Nancy and Ed Leeper, "Fetal Loss Associated With Two Seasonal Sources of Electromagnetic Field Exposure", Am. J. of Epidemiology, Vol. 129, No. 1, 1989

Home Heating With Ceiling Electric Heat As A Cause of Spontaneous Abortion - Comparison With Previous Study of Electric Blankets and Water Beds



Abortions related to change in temperature

Chart 2

Popular Tranquilizer Diazepam (VALIUM) Causes Learning Disabilities and Hyperactivity in Laboratory Animals 9 *(See also 31d)*

A recently published study by Frieder, Epstein and Grimm found that exposure of rats to 10 parts per million of Valium during pregnancy or lactation caused learning disabilities and hyperactivity in the offspring.

"Exposure during mid-gestation resulted in early and transient hyperactivity, but no learning or memory deficits at 2 months of age were observed. However, both late prenatal and early postnatal exposure to diazepam resulted in significant behavioral changes. Late prenatal treatment caused no hyperactivity but resulted in poor performance on the learning and retention of a choice discrimination taks, while early postnatal exposure resulted in consistent and lasting hyperactivity and in substantial discrimination learning and retention deficits at 2 months of age."

The effects of prenatal exposure to drugs like Valium in the benzodiazepine family on learning and behavior appear to include first a "floppy infant syndrome" which is related to continued presence of the drug in the body, and some longer-range effects on behavior which include hyperactivity and learning disabilities. It is believe that one reason for this is interaction of the drugs with the benzodiazepine receptors in the brain.

"Benzodiazepine receptors in rates were shown to develop rapidly from about embryonic day 16. At this time the receptors are about 2%, at birth 25% of the adult levels, which they reach only at 21 days after birth. This would mean that during the 2nd week of gestation in this study...there would be no specific binding of DZP to brain membranes, while during the 3rd weeks of gestation...or postnatally, substantial receptor binding could occur. A number of studies, both in rats and on human newborns and premature infants, show that the capacity of the fetus or newborn to dispose of the drug is very low. In the newborn hydroxylated compounds appear in urine only after 8-10 days of age, showing that the metabolism of DZP at this time is very slow..."

It was suggested that the long persistence of the drug interfered with the proper development of the neurotransmitter mechanisms important for activity, learning and memory.

DOCTOR PRESCRIBED VALIUM AND LIBRIUM AND BIRTH DEFECTS
Tranquilizers Also Cause Congenital Malformations. Smoking Any Cigarettes Compounds The Problem - Study of Connecticut Births 3

Table 8. Association of Exposure to Tranquilizers and Cigarettes in Pregnancy with Congenital Malformations in Offspring

Daily no. of cigarettes*	Use of tranquilizers†	Case	Control	Odds ratio	1-tailed Fisher exact test
None	No	848	1916	1.44 ←	.079
	Yes	30	47		
1-20	No	361	836	3.70 ←	.003
	Yes	21	13		
21+	No	75	122	3.70 ←	.000
	Yes	18	8		

Test for 3-factor interaction: $G_3^2 = 6.715$, $P < .05$.

Test for differential effect of tranquilizers between nonsmokers and all smokers: $\Delta G_1^2 = 6.715$, $P < .01$.

* Based on cigarettes smoked daily during the third month of pregnancy.

† Exposure only in the first two trimesters.

Source: Bracken & Holford, Obstetrics and Gynecology, September 1981
Frieder et al, Psychopharmacology, Vol. 83, 1984

ASPIRIN AS DANGEROUS AS LEAD!

Aspirin:

USE DURING PREGNANCY CAN SHARPLY REDUCE THE IQ OF THE CHILD

A just published study of 421 children, 4 years old, whose mothers had used either aspirin or acetaminophen during pregnancy to alleviate symptoms like headache, found a profound loss of IQ and an increase in attention decrements among the children exposed to aspirin in utero. This was not true of acetaminophen.

Indeed, in the unadjusted scores, use of aspirin several times a week or more during pregnancy caused an 8 point drop in IQ, or a 7.1 percent loss. About 13 percent of all women were found to have taken aspirin during pregnancy several or more times a week. 45 percent of pregnant women used some aspirin.

TABLE 2. Mean child IQ scores by mother's education
Unadjusted scores (N = 421)

	Aspirin		Percent IQ Loss
	Less than several a wk	Several times a wk or more	
College graduates	120 (104)ª	112 (7)	-6.7%
High-school graduates	111 (215)	103 (44)	-7.2%
Not high-school graduates	95 (48)	92 (3)	-3.1%
Column means	113 (367)	104 (54)	-7.1%

ªSample size is in parentheses.

This relationship stood up after controlling for a wide range of other variables. It was found that there was a sex difference, with girl children losing 10.1 points in mean IQ, and boys 2.3 points when their mothers took several or more aspirins per weeks during pregnancy.

How It May Work

Unlike some agents, aspirin does not affect head size. It appears to work by causing bleeding in the brain of the fetus, or by affecting critical timing in brain development. A significant increase in children's attention deficits was also found, after correcting for a wide range of other variables.

Aspirin has also been linked to a range of central nervous system anomalies, major and minor malformations and oral clefts by several studies.

Source: Streissguth, Ann, Pytkowics et al, "Aspirin and Acetaminophen Use by Pregnant Women and Subsequent Child IQ and Attention Decrements", Teratology, Vol. 35, (1987) 14

PHENOBARBITAL

Jacqueline Farwell and collegues reported in the February 8, 1990, New England Journal of Medicine that the use of the drug phenobarbital to control febrile seizures in children under the age of 3 was not only ineffective in preventing the progression to epilepsy, but also reduced the intelligence of the children by 8.4 IQ points.

This was a shocking result. And the IQ change appears to be permanent. Six months later, the mean IQ of those exposed children was still 5.2 IQ points lower than in the controls.

able 3. Average Stanford–Binet IQ Scores at the Final Visit, According to Expected IQ Level and Treatment Group.

xpected IQ Level	Phenobarbital Group			Placebo Group		
	No. at Base Line	No. at Final Visit	Average IQ	No. at Base Line	No. at Final Visit	Average IQ
	24	13	91.38	19	13	102.69
	17	11	101.09	26	21	98.81
	22	17	98.59	21	19	104.74
	21	17	104.77	22	20	109.25
	24	20	114.05	21	19	117.37
otal	108	78	—	109	92	—
orvitz–Thompson average*	—	—	100.94	—	—	106.17

*According to the Horvitz–Thompson averages, the difference in IQ scores was 5.23 in favor the placebo group, and the t statistic was 2.005 (P adjusted for three interim analyses, 0.052).

The Network's Petition of 1985

Four years ago, we petitioned the then Secretary of HHS, Margaret Heckler and also the Food and Drug Administration for a limitation on phenobarbital use.

Phenobarbital has been a very popular drug among obstetricians in the past. The Collaborative study found that about 10 percent of women were given barbituates (phenobarbital is one) during pregnancy in the 1960's, and it was associated with a much elevated rate of birth defects and learning disabilities in the children.

Research at the National Institute of Health had also found that exposure of laboratory animals to phenobarbital during pregnancy led to infertility of the offspring. This finding has been since replicated by laboratories around the world.

Most uses of phenobarbital during pregnancy are not necessary. As Reynolds put it, "Barbituates were once given in large doses to women in labour, on the principle that if pain cannot be relieved, the sufferer can at least be kept quiet". Similar considerations were cited by doctors who prescribed the drug during the pregnancy itself.

Secretary Heckler and the Food and Drug Administration rejected every proposal of our petition in 1985, including a refusal to stop paying for unnecessary prescriptions of phenobarbital through Medicaid.

WE PETITION HHS AND FDA

The Network submitted a petition to Dr. Louis Sullivan, Secretary of the Department of Health and Human Services and to the Food and Drug Administration on April 30, 1990.

We asked for a ban on all uses of phenobarbital except for clear cases of epilepsy, during pregnancy, lactation and for children under the age of 3.

If you would like a copy of the petitions, send a self-addressed stamped envelope.

Who To Write

If you would like to write a letter in support of the petition, it would probably be best addressed to:

Dr. Louis Sullivan
Secretary
Department of Health and Human Services
Hubert Humphrey Building
200 Independence Ave. S.W.
Washington, D.C. 20201

Source:

Farwell, Jacqueline, R. et al, "Phenobarbital for febrile seizures- effects on intelligence and on seizure recurrence", New Eng. J. of Medicine, Feb. 8, 1990

See Petition on Drugs, 1985, for more information on literature - order form

HOME PESTICIDES & CHILD HEALTH

Ten years ago, the Environmental Protection Agency surveyed American households, finding that 91 percent of all households use pesticides in and around the home.

This broke down into the following:

- 84 percent used pesticides in the home, with considerable regional variation;
- 21 percent used pesticides in the garden;
- 39 percent used pesticides in the yard.

These figures come from the 1976-7 period, and we can expect that yard and garden use has probably significantly increased in the past ten years, as commercialization of lawn service has become popular.

It was found that people were suffering some acute damage because of this. About 3 percent of all users complained of pesticide poisoning, including dizziness, headache, nausea or vomiting. It was also estimated that 2 percent of all households had suffered an economic loss due to pesticide use, such as destruction of desirable plants, staining of furniture or carpets, injury to pets, or loss of cash crops or other cash items.

Little survey work has been done on the chronic health effects of home pesticide use, though two recent studies indicate that home pesticide use may be more hazardous to children than had been expected.

HOME PESTICIDES AND CHILDHOOD CANCER

A 1987 study by Lowengart and collegues discovered that household and garden pesticides posed severe risks of leukemia in children, as is noted by the table. While leukemia is a relatively rare disease, it indicates damage to the immune system that can produce a wide range of other ailments, including infections, and other types of cancer.

Unfortunately, the E.P.A. study of use does not permit us to estimate the frequency of home use of pesticides, that would permit us to calculate the percentage of leukemias that are produced by home use of pesticides.

It does appear that home pesticide use may in fact account for a significant percent of total annual leukemias in children.

HOME PESTICIDE USE AND BIRTH DEFECTS

A number of families in the Coffs Harbor area of New South Wales in Australia complained that the use of a relatively new pesticide TILT, for banana spraying was causing a high rate of birth defects in the region.

This pesticide is being used for the dipping of seed for sugar cane in Hawaii, and the Environmental Protection Agency is considering extending its use substantially, even though laboratory tests show that it also causes cancer.

CHILDHOOD LEUKEMIA AND HOME PESTICIDE USE

Factor	Odds Ratio for Leukemia	One-Sided p Value
Home Exposures to either parent during pregnancy, nursing, and fathers during pregnancy		
Household pesticides - once/week or more	3.8	.004
Garden pesticides or herbicides - once/month	6.5	.007
Burned incense during pregnancy - once/week or more	2.7	.007
Paint laquer exposure to mother - once/week or more	1.8	.03

The Department of Health of New South Wales employed the School of Public Health and Tropical Medicine of the University of Sydney to investigate these congenital malformations.

From this study, we have one of the first estimates of a strong contribution of home pesticide use to human birth defects. The brands of home pesticides were not disclosed by the report, however.

Most of the outdoor pesticide (occupational) exposure was to mothers who worked at home, and presumably grew produce for sale. See the table below.

E.P.A. STRIKES A DEAL WITH VELSICOL TO STOP MANUFACTURING CHLORDANE

One of the more potent leukemia causing pesticides is the termite poison, chlordane, which was banned for most agricultural uses in 1980 because it caused cancer.

On August 11, 1987, the Environmental Protection Agency announced an agreement with the manufacturer of chlordane and the related heptachlor, that stops the production of the chemicals, but allows the sale of existing stocks held by distributors and pest control operators, even though it has been estimated that this further use will result in 1,300 more American cancers.

It comes as welcome news the removal of these chemicals from the market, that not only have been linked to childhood cancers, but which may also cause learning disabilities and which contaminate human breast milk.

The National Coalition Against the Misuse of Pesticides and nine other groups filed suit in federal court in July to try to prevent the sale of the remaining stocks. This lawsuit succeeded. In February, the Federal District Court for the District of Columbia ruled that E.P.A. had violated the law with this voluntary agreement, and ordered the end of sales of stocks of chlordane and heptachlor.

**CLEFT LIP AND PALATE CASES AND HOME PESTICIDE USE

Summary of odds ratios of exposures either before or during the first three months of pregnancy among cases and controls

Exposures	Number of Cases (18)		Number of Controls (50)		ODDS RATIO
	Exp.	Not Exp.	Exp.	Not Exp.	1.0= normal
Maternal exposures before pregnancy					
-outdoor pesticides	11	7	17	33	3.1
-outdoor pesticides (occupational)	6	12	5	45	4.5
-indoor pesticides (excl. fly-sprays)	4	14	2	48	6.9
-indoor pesticides (incl. fly-sprays)	14	4	35	15	1.5
-water supply (town/other)	13	5	40	10	0.7
Maternal exposure in first three months of pregnancy					
-chemical cleaning agents	7	11	24	26	0.7
-paints	2	16	16	34	0.3
-oil-based or varnish paints	1	17	8	42	0.3
-paint remover, thinner or solvent	2	16	6	44	0.9
-glues and adhesives	0	18	7	43	-
-outdoor pesticides	6	12	7	43	3.1
-outdoor pesticides (occupational)	6	12	6	44	3.7

Sources: Lowengart, Ruth, A. et al, "Childhood Leukemia and Parents' Occupational and Home Exposures" Journal of Nat. Cancer Inst., Vol. 79, No. 1, July 1987

Savage, Eldon et al, National Household Pesticide Usage Study, 1976-7, Environmental Protection Agency, EPA 540/9-80-002, July 1980

Lancaster, Paul and Jennifer Baker, Report on the Incidence of Major Congenital Malformations in the Coffs Harbor Region of New South Wales, National Perinatal Statistics Unit, School of Public Health and Tropical Medicine, University of Sydney for the New South Wales Department of Health, Australia, Dec. 1985

PESTICIDE EXPOSURE AT HOME OR ON THE JOB DAMAGES PREGNANCY

Very few studies are available concerning the effects of pesticides in the home and pregnancy. We reviewed two in a previous newsletter, showing rather large adverse effects on birth defect and childhood cancer rates.

Savitz and colleagues recently reported on self-reported exposure to pesticides and pregnancy. It was found that maternal exposure to pesticides either at home or work was associated with a 30 to 60 percent increased risk of stillbirth. Exposure to the father was associated with increased stillbirth and a small-for-gestational age child.

__Source:__
Savitz, David, A. et al, "Self-reported exposure to pesticides and radiation related to pregnancy outcome.." Public Health Reports, Sept./Oct. 1989, Vol. 104, No. 5

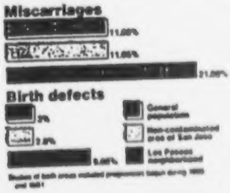
NEWS ON TOXICS AND BIRTH DEFECTS

LEAKING SOLVENT TANK IN THE SILICON VALLEY CAUSES TRIPLING OF BIRTH DEFECTS

We reported in the last newsletter on the study of birth defects in Woburn, Massachusetts caused by solvents in the drinking water from a toxic waste dump.

Another case of solvent produced birth defects involving ground water contamination has developed in the Silicon Valley. A leaking storage tank of the Fairchild Camera Company released the solvent TCA (1,1,1 trichloroethane) into the ground water.

Families using the wells contaminated with TCA experienced a doubling of miscarriages and a three time increase in birth defects.



Heart defects were particularly noted. Laboratory studies show that heart defects are produced in animal offspring when exposed to TCA during pregnancy.

For a copy of the findings of the California study, you would want to write the address below and request, "Pregnancy Outcomes in Santa Clara County, 1980-1982, Summaries of Two Epidemiological Studies":

Calif. Dept. of Health Services
2151 Berkeley Way
Berkeley, California 94704

San Francisco Examiner,

Table 1

BIRTH DEFECTS ASSOCIATED WITH DRINKING WELL WATER CONTAMINATED BY SOLVENTS FROM A TOXIC WASTE DUMP

Unfavorable Health Event	Contaminated Wells G/H	Pine Street	Sweetwater Brook
Leukemia	Yes	*	*
Perinatal Death	Yes	No	No
Birth Defects			
Eye/Ear	Yes	No[1]	No[1]
Environment Related	Yes	No[1]	No
Childhood Disorders			
Lung/Respiratory	Yes	Yes	No
Kidney/Other Urinary	Yes	No	No
Allergies/Skin	No	Yes	No
Neurologic/Sensory	No	No	Yes

* Leukemia analysis not carried out.

1. Relationship explained by access to wells G/H.

THE WOBURN, MASSACHUSETTS STUDY OF PEOPLE DRINKING WATER CONTAMINATED BY A TOXIC WASTE DUMP

Another landmark study on birth outcomes relating to toxic substances exposures was recently completed by the School of Public Health of Harvard University. The study was prompted by a cluster of childhood leukemia in a Massachusetts town.

High rates of infant mortality and a variety of birth defects was found to be associated with drinking water from wells contaminated with some common industrial solvents such as trichloroethylene. This is an interesting study because these solvents are found at most dump sites in the nation.

Table 1 summarizes the findings of the Woburn study. You can get a copy of a summary of the study from, School of Public Health, Harvard University, 677 Huntington Ave., Boston, Mass. 02115. Full report $10.00 Feb. 1984

CALIFORNIA STATE COMMISSION FINDS THAT TOXIC CHEMICAL CONTAMINATION COSTS THE STATE $4 BILLION

A June 1985 report of the California Commission for Economic Development concluded that five economic costs associated with toxic contamination: cleanup costs, regulatory costs, litigation costs, health care costs, and resource losses, added up to a $4 billion a year bill for California.

It was recommended that policies be structured to bring down costs in the future. *From: Poisoning Prosperity, The Impact of Toxics on California's Economy, $5, to Calif. Commission on Economic Develop., Office of Lieutenant Governor, State Capitol, Room 1028, Sacramento, Calif. 95814*

BREAST MILK CONTAMINATION 20

HAWAII STUDY SHOWS THAT BREAST MILK CONTAMINATION WITH THE PESTICIDE HEPTACHLOR CAN LEAD TO DELAY IN BRAIN DEVELOPMENT UP TO 8 MONTHS AFTER BIRTH

Jeanne Hoffman of the University of Hawaii completed her doctoral study of 120 infants exposed to human milk which was seriously contaminated with the pesticide heptachlor.

This study is of interest to mainlanders because, while the pesticide was used only on pineapples, it also contaminates the widely use termite poison, chlordane. It is also very likely that chlordane itself has similar impacts on infants.

Hoffman found several impacts:

1. There were higher rates of physical birth defects in the most exposed infants, suggesting an adverse effect of heptachlor during pregnancy itself.

2. Moderate heptachlor levels in the breast milk were associated with significantly lower scores on the Bayley Infant Scales at 4 and at 8 months, a period where the brain development continues rapidly in infants.

3. In the lightly exposed cases, the breast milk proved sufficiently superior to bottle milk to counter the adverse effects of the pesticide in terms of test scores.

4. Significant physical effects were also noted in these infants, including lower birth weight, smaller head circumference, and presence of jaundice.

A follow-up study is needed to determine some years in the future whether these delays in brain development translate into learning disabilities.

Source: Hoffman, Jeanne, S., "The Effects of Prenatal Heptachlor Exposure on Infant Development", Dissertation to the University of Hawaii towards a Degree of Doctor of Philosophy in Psychology, May 1985

NEW MICHIGAN STUDY INDICATES THAT PCB's EXPOSURE DURING PREGNANCY CAN CAUSE A DELAY OF BRAIN DEVELOPMENT OF THE INFANT UP TO 7 MONTHS OF AGE

In the March 1985 petition on breast milk purity, we described a study from Michigan of 313 infants, some of whose mothers ate fish from Lake Michigan known to be contaminated with the industrial fire retardant PCB's.

Severe adverse effects were seen on the infants among those mothers eating only 2 to 3 fish per month.

A just published follow-up study of 123 of these infants at seven months of age found that significant brain development delay had occurred in the infants most exposed during pregnancy. In other words, by the time one begins to wonder whether it is safe to breast feed the child, it is already too late!

As the chart below indicates, PCB's in umbilical cord serum at birth had a significant correlation with responses of 81 infants 7 months later on the fixation to novelty test.

This test has a good correlation with future verbal IQ . For example, correl-

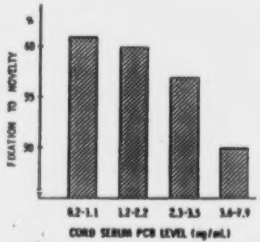


FIG. 1.—Dose-response relationship of visual recognition memory with cord serum PCB level after adjusting for potential confounding variables, F(3,74) = 3.90, p = .01. Group N's are 20, 21, 20, and 20, respectively.

ations between this infant visual recognition score and later vocabulary tests of IQ in four samples averaged .47. Rose and Wallace have also demonstrated similar correlations with IQ scores among preterms from 34 months through 6 years of age.

Source: Jacobson, Sandra W. et al, "The Effect of Interauterine PCB Exposure on Visual Recognition Memory", Child Development, Vol. 56, 1985

TERMITE POISONS AND BREAST MILK

Two recently published studies from Australia find that treatment of the home for termites with the chlorinated pesticides like chlordane, aldrin, and dieldrin can produce significant breast milk contamination for a pregnant woman living in that home.

The authors conclude, "Thus, although not conclusively proving that the use of aldrin and dieldrin in the protection of homes against termites is a major source of contribution to the high levels of dieldrin in human milk in Western Australia, the evidence strongly supports this theory..."

A second study compared termite applications with resulting breast milk contamination in 14 cases. The table below for Donor 3 is a typical example of their findings.

Chlordane Levels in Donor No 3. (Values Expressed in ng/g Whole-milk Basis)

Sample No.	Sampling time relative to treatment	Chlordane
1.	3 days prior	Tr
2.	1 week after	63
3.	3 weeks after	66
4.	7 weeks after	64
5.	11 weeks after	26
6.	15 weeks after	3
7.	17 weeks after	2

Tr = trace less than 1 ng/g

In the case of dieldrin, it was found that breast milk levels in some cases exceeded the recommended adult dosages in the diet of the World Health Organization by up to 14 times. One could expect learning disabilities in the children as a result, and possible childhood cancers. See article on heptachlor, for example.

The states of New York, Massachusetts, and Rhode Island have proposed or initiated bans on the use of chlorinated termite poisons. Hasn't the time come for the Environmental Protection Agency to take similar action, in view of the fact that good alternatives exist?

Source: Stacey, Conway, I. et al, " Organochlorine Pesticide Residue Levels in Human Milk..." Archives of Environ. Health, March/April 1985 And Stacey and Tatum, "House Treatment with Organochlorine Pesticides and Their Levels in Human Milk...", Bull. Environ. Contam. and Tox., Vol. 35, 1985

FIRST ACTION ON HEXACHLOROBENZENE

With the August newsletter, we noted that the Environmental Protection Agency was undertaking a coordinated study and action program with other federal agenci on the chemical HCB, hexachlorobenzene, which is commonly found in breast milk.

HCB is manufacturered in Argentina today as a fungicide for wheat seed, but has never been registered in the United States for this purpose. The major sourc of American exposure appears to be contaminated pesticides and industrial uses as in the making of rubber and aluminum. It is estimated that 8 and one half million pounds of HCB wastes are produced annually in the United States.

In March 1985, E.P.A. took a first action to deal with contaminated pesticides by limiting HCB content in the herbicide picloram to 200 parts per million. (Picloram is better known to many of you as Agent White of the Vietnam War, and is still widely used in the United States. Many have urged a ban of the chemical itself.)

Many more action like this will be needed to bring HCB levels down in the United States. But it is a promising start.

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DIOXIN IN BREAST MILK

Dr. Arnold Schecter, professor of preventative medicine at the State University of New York, released his findings about dioxins in human breast milk in New York State at a press conference with the Environmental Defense Fund in December 1987. This study, has been published by Chemosphere.

Average total dioxin levels, standardized for the TCDD dioxin which is the most toxic, was a shocking 1.04 parts per trillion. Some mothers average 4.72 parts per trillion, which would give cancer to 50 percent of laboratory rats, if fed as a complete milk diet as is a baby. The tables below summarize the comparison of New York breast milk dioxin levels and laboratory findings.

New York Mothers' Milk Dioxin Enough To Give 50% Cancer To Laboratory Rats?!

Dioxins in Human Milk (New York State)	(Whole Milk Basis) Average Levels	High Averages
TCDD	.19 ppt	.38 ppt
Total dioxins and furans, standardized for TCDD toxicity, (toxic equivalency factor)	1.04 ppt	4.72 ppt

Table 2. Summary of Neoplastic Alterations Observed in Rats Fed Subacute Levels of (TCDD), 2,3,7,8-tetrachlorodibenzo-p-dioxin for 78 Weeks. (J.P. Miller et al, Univ. of Wisconsin)

Level of TCDD	No. of Animals With Neoplasms	Percent With Cancers	No. of Cancer Types
0	0	0 percent	0
1 ppt	0	0 percent	0
5 ppt	5	50 percent	6
50 ppt	3	30 percent	3
500 ppt	4	40 percent	4
1 ppb	4	40 percent	5
5 ppb	7	70 percent	10

WILL DIOXINS CAUSE LEARNING DISABILITIES?

Recent studies note that TCDD dioxin has a very similar chemical structure to thyroid hormones, and may indeed share the same receptors. Interference with thyroid hormones during pregnancy will produce learning disabilities.

Furthermore, there is reason to believe that chemicals that induce the liver enzymes like dioxin also cause learning disabilities. For these two reasons, TCDD dioxin would have to rank high in a suspect list as a learning disability cause.
See: Evans, R.W., Science, May 13, 1988, Bombick, D.W. et al, Proc. Nat. Acad. Sci., p. 4128, June 88

Dioxins come from numerous sources such as municipal trash incinerators, the wood preservative pentachlorophenol, and the paper industry. Recently, Greenpeace published a report on dioxins from paper manufacture and in the paper itself, No Margin of Safety. The use of chlorine for the bleaching of paper and pulp is the source of the dioxin. It is a problem that can be easily corrected by simply substituting oxygen bleaches for chlorine as is done in Europe.

Opposition to municipal trash incinerators is widespread in the United States now. The plastics in the trash are the source of much of the dioxins from the burning process, which contaminate farm animal products due to dioxin in the pastures, as well as contaminating fish. Waste Not is a good publication summarizing weekly what is happening in the citizen effort to defeat municipal trash incinerators.

Source: Press release, Environmental Defense Fund, Dec. 17, 1987; J.P. Van Miller et al, "Increased Incidence of Neoplasms in Rats Exposed to Low Levels of 2,3,7,8-Tetrachlorobenzo-p-Dioxin", 1978? (U. of Wisc., Dept. Pathol. and Region. Primate Center); Van Strum, Carol and Paul Merrell, No Margin of Safety, 1987, $10 from Greenpeace Great Lakes Toxics Campaign, 427 Bloor St. W., Toronto, Ontario M6S 1X7; Waste Not, publication of Work on Waste, 82 Judson, Canton, New York 13617, $25/year, $15 for Students and

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Table 2

WORK ENVIRONMENT PRODUCES SPONTANEOUS ABORTIONS

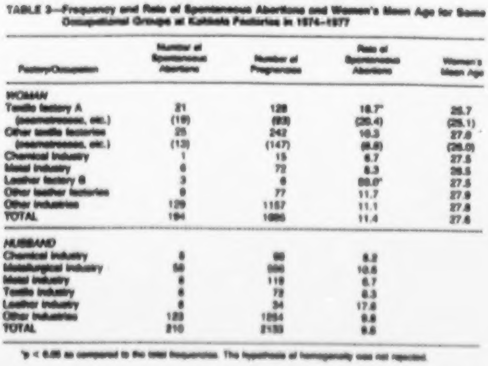


TABLE 3—Frequency and Rate of Spontaneous Abortions and Women's Mean Age for Some Occupational Groups at Kuhlavla Factories in 1974–1977

Factory/Occupation	Number of Spontaneous Abortions	Number of Pregnancies	Rate of Spontaneous Abortions	Women's Mean Age
WOMAN				
Textile factory A	21	126	16.7*	26.7
(seamstresses, etc.)	(19)	(93)	(20.4)	(26.1)
Other textile factories	25	242	10.3	27.8
(seamstresses, etc.)	(13)	(147)	(8.8)	(26.0)
Chemical industry	1	15	6.7	27.5
Metal industry	6	72	8.3	26.5
Leather factory B	3	6	50.0*	27.5
Other leather factories	9	77	11.7	27.9
Other industries	129	1157	11.1	27.8
TOTAL	194	1695	11.4	27.6
HUSBAND				
Chemical industry	5	61	8.2	
Metallurgical industry	58	546	10.6	
Metal industry	8	119	6.7	
Textile industry	6	72	8.3	
Leather industry	6	34	17.6	
Other industries	123	1254	9.8	
TOTAL	210	2133	9.8	

*p < 0.05 as compared to the total frequencies. The hypothesis of homogeneity was not rejected.

MISCARRIAGES OF PREGNANCY IN FINLAND CAUSED BY EMPLOYMENT OF HUSBAND AND WIFE

There is an impressive study of pregnancy outcome among workers in Finland, using birth certificates. It was found that the occupation of either the husband or the wife can produce higher rates of spontaneous abortions in their pregnancies.

While women who worked in industrial settings generally had higher rates of miscarriage than inactive women, rates were much higher in some industries such as textiles and leather. For the men, much higher miscarriage rates were found when they worked in metallurgy or leather.

Particular plants had particularly high rates of miscarried pregnancies, and there was a synergistic effect, where the husband worked on one plant and the wife in another.

For example, when the wife worked in Factory A, and the husband worked in a metallurgical plant, 29 percent of all pregnancies were miscarried.

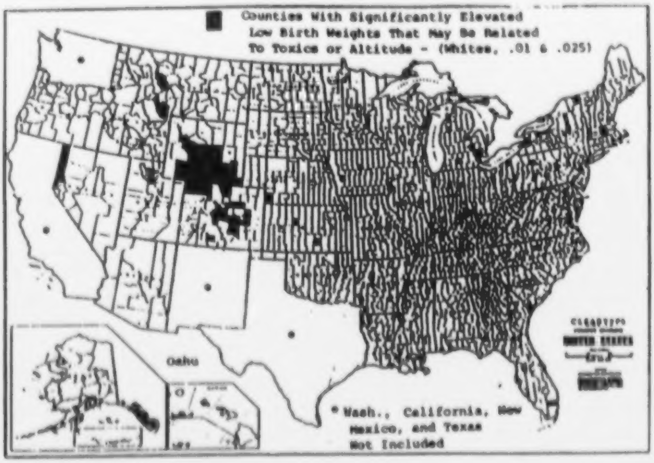
Table 2 summarizes some of the results of this study. A copy can be gotten from:

American Journal of Public Health
January 1983, Vol. 73, No. 1

NEWS ON TOXICS AND BIRTH DEFECTS

Map 1

TOXICS AND LOW BIRTH WEIGHTS
BY COUNTY - Find Your County



▪ Counties With Significantly Elevated Low Birth Weights That May Be Related To Toxics or Altitude - (Whites, .01 & .025)

* Wash., California, New Mexico, and Texas Not Included

LANDMARK STUDY BY THE ENVIRONMENTAL PROTECTION AGENCY ON LOW BIRTH WEIGHTS AND TOXICS

It has long been known that poor nutrition and poor prenatal care can produce high rates of low birth weights. Exposures to toxics is another important factor in low birth weights. For example, cigarettes smoked during pregnancy have long been linked to low birth weights.

A recent study from the Environmental Protection Agency clarifies the relationship between nutrition, prenatal care, and toxics exposures. This landmark study of low birth weights by county produced the map above - showing areas where toxic exposures may be producing statistically significant high rates of low birth weights.

(to next page)

E.P.A. surveyed approximately 2 million birth certificates - excluding those of California, Texas, New Mexico, and Washington where the certificates did not present sufficient information. One finding was that every region of the nation has at least one high-risk county where high rates of low birth weights seem associated with socio-economic factors.

Screening for Environmental Causes

Removal of those cases of low birth weights associated with socio-economic factors left a residual or "unexplained" of high rate counties. These are presented on Map 1. In those counties, exposures to toxic substances or to the low oxygen levels of high altitude - as in the Rocky Mountains - appear to be important and likely causes of the low birth weights.

Conclusion

The study concluded"

"Clusters in the Rocky Mountain region and in certain Northern industrialized states suggest the strong influence of environmental factors such as altitude, mineral extraction industries (eg. lead, uranium, silver mining), heavy industry (steel, automobile, chemical) or perhaps agricultural spraying.

We can provide you a copy.

Glick, Barry, J., Anne K. Welsh and Casey L. Jason, "The Geographical Distribution of Unexplained Low Birth Weight", Study financed by the Environmental Protection Agency, 1983?

COMMENT OF THOMAS H. JUKES

Comment on testimony by Nancy Greenspan, September 13.

Nancy Greenspan is understandably apprehensive about the health of her daughter Sarah, with juvenile diabetes. However, she may have drawn wrong conclusions about the effects of meat and ammonia on blood glucose.

Meat does not contain "large amounts of growth hormones and antibiotics." A withdrawal period is used when meat animals receive antibiotics, so that there are no residues present in the meat. Antibiotic residues are also destroyed by cooking. The only growth hormones in meat are those that are produced naturally in the body of animals, including "organic chicken." There is no difference between commercially produced meat and "organic" meat or chicken.

Ammonia is present in normal blood. It is formed by the metabolism of protein in the body. Ammonia has a strong odor, but it is not dangerous in low concentrations. The odor of ammonia is very pungent, and it is used as smelling salts (ammonium carbonate) to revive people who have fainted. It is unlikely that the odor of ammonia was responsible for Sarah's increase in glucose levels after dinner. It is more likely to have been coincidental. Only a controlled test could establish this.

The fluctuations in Sarah's blood glucose between the ages of 2 and 3 1/2, when she was first placed on insulin are probably due in part to a deficiency of insulin.

THOMAS H. JUKES

[illegible]

U.S. House of Representatives
SELECT COMMITTEE ON
CHILDREN, YOUTH, AND FAMILIES
385 House Office Building Annex 2
Washington, DC 20515

[illegible]

October 3, 1990

Susan Pollack, M.D., Instructor
Occupational Medicine and Pediatrics
Box 1057
Mount Sinai School of Medicine
1 Gustave L. Levy Place
New York, NY 10029

Dear Dr. Pollack:

I want to express my personal appreciation to you for appearing before the Select Committee on Children, Youth, and Families at our hearing, "Environmental Toxins and Children: Exploring the Risks," Part II, held here in Washington on September 13. Your testimony was, indeed, important to our work.

The Committee is now in the process of preparing the transcript for printing. It would be helpful if you would go over the enclosed copy of your remarks to assure that they are accurate, and return the transcript to us by October 10 with any necessary corrections.

Also, as requested at the hearing, the committee would also appreciate an update of your written statement to reflect your oral testimony.

Let me again express my thanks, and that of the other members of the Committee, for your participation.

Sincerely,

George Miller

GEORGE MILLER
Chairman
Select Committee on Children,
Youth, and Families

Enclosure

No further communication received by time of printing.

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